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SEARCH REQUEST FORM

65610

Requestor's Name: P. Krwiecek

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Art Unit: 1614

Search Topic: 12/15/00

Please write a detailed statement of search topic. Describe specifically as possible the subject matter to be searched. Define any terms that may have a special meaning. Give examples or relevant citations, authors keywords, etc., if known. For sequences, please attach a copy of the sequence. You may include a copy of the broadest and/or most relevant claim(s).

Please search:
methods of treating cerebral ischemic conditions, (L30)
such as thrombo-embolic (L31), vascular spasm (L32), cardiac
dysfunction (L33), cardiopulmonary bypass (L34), comprising
administering gamma-
or beta- tocopherol
or delta-

not alpha or gamma-CEHC

Thanks

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Date completed:
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Elapsed time: _____
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Type of Search
_____ N.A. Sequence
_____ A.A. Sequence
_____ Structure
_____ Bibliographic

Vendors
_____ IG Suite
_____ STN
_____ Dialog
_____ APS
_____ Geninfo
_____ SDC
_____ DARC/Questel
_____ Other

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L28 9558 SEA FILE=HCAPLUS ABB=ON PLU=ON BRAIN?/CT(L)ISCHEMIA?
L29 10801 SEA FILE=HCAPLUS ABB=ON PLU=ON ("BRAIN, DISEASE (L) ISCHEMIA" /CT OR "BRAIN (L) ISCHEMIA"/CT OR "CEREBRAL ISCHEMIA"/CT OR "GLOBAL CEREBRAL ISCHEMIA"/CT OR "ISCHEMIC BRAIN"/CT OR "ISCHEMIC CEREBROVASCULAR DISEASE"/CT)
L30 10852 SEA FILE=HCAPLUS ABB=ON PLU=ON L28 OR L29
L31 899 SEA FILE=HCAPLUS ABB=ON PLU=ON ("EMBOLISM (L) THROMBOEMBOLISM "/CT OR "EMBOLISM (L) THROMBO-"/CT OR "VEIN (L) DISEASE, THROMBOEMBOLISM"/CT OR THROMBOEMBOLISM/CT OR "THROMBOEMBOLISM VEIN"/CT OR "VENOUS THROMBOEMBOLISM"/CT)
L32 385 SEA FILE=HCAPLUS ABB=ON PLU=ON ("BLOOD VESSEL, DISEASE (L) SPASM"/CT OR "BLOOD VESSEL (L) SPASM"/CT OR "SPASM BLOOD VESSEL"/CT OR "VASCULAR SPASM"/CT OR VASOSPASM/CT OR "VASOSPAST IC DISORDER"/CT)
L33 24988 SEA FILE=HCAPLUS ABB=ON PLU=ON "HEART, DISEASE"/CT
L34 728 SEA FILE=HCAPLUS ABB=ON PLU=ON ("CARDIOPULMONARY BYPASS"/CT OR "CIRCULATION (L) EXTRACORPOREAL, CARDIOPULMONARY BYPASS"/CT)
L35 37188 SEA FILE=HCAPLUS ABB=ON PLU=ON L30 OR L31 OR L32 OR L33 OR L34
L37 76505 SEA FILE=HCAPLUS ABB=ON PLU=ON L35 OR (BRAIN OR CEREBRAL)(2A) ISCHEM? OR THROMBOEMBOL? OR (VASCULAR OR BLOOD(W)VESSEL? OR ARTER? OR VEIN)(2A)SPASM? OR CARDIAC(W)DYSFUNC? OR HEART(2A)DIS EAS? OR CARDIOPULMONAR?(2A)BYPASS
L44 3 SEA FILE=REGISTRY ABB=ON PLU=ON 7616-22-0 OR 148-03-8 OR 119-13-1
L45 3007 SEA FILE=HCAPLUS ABB=ON PLU=ON L44 OR (GAMMA OR BETA OR DELTA)(W)TOCOPHEROL? OR GAMMA(W)CEHC
L46 32 SEA FILE=HCAPLUS ABB=ON PLU=ON L45 AND L37

L46 ANSWER 1 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 2002:274500 HCAPLUS
TI Association of serum antioxidant capacity with coronary artery disease in middle-aged men
AU Nojiri, Shuko; Daida, Hiroyuki; Mokuno, Hiroshi; Iwama, Yoshitaka; Mae, Kiyoshi; Ushio, Fusao; Ueki, Takato
CS Tama Branch, Tokyo Metropolitan Research Laboratory of Public Health, Tokyo, 190-0023, Japan
SO Japanese Heart Journal (2001), 42(6), 677-690
CODEN: JHEJAR; ISSN: 0021-4868
PB Japanese Heart Journal Association
DT Journal
LA English
AB The possible involvement of oxidative damage in the progression of atherosclerosis has been suggested. There is some evidence that antioxidant therapy may be beneficial for the prevention of coronary **heart disease**. In this study, we investigated the relationship between coronary artery disease (CAD) and serum antioxidative status by measuring the total antioxidant status (TAS). Other relevant antioxidants, such as retinol, .alpha., .**gamma**.-**tocopherol**, ascorbic acid, .alpha., .beta.-carotenoids, erythrocyte glutathione peroxidase (GSH-Px) and oxidative products, were also detd. in 31 male CAD patients with angiog. defined CAD and 66 male controls, aged 40-70 yr, in a case-control study. The TAS levels, ratio and the concns. of retinol, albumin, total protein and HDL cholesterol were significantly lower in the CAD patients than in the controls ($p<0.01$), and .alpha.-tocopherol and .alpha./.**gamma**.-**tocopherol** were significantly higher in the CAD patients than in the controls. The TAS level correlated pos. with .gamma.-GTP, GPT, GOT and uric acid ($p<0.01$). A multiple regression anal. in the CAD patients revealed that the TAS levels correlated most neg. with the no. of diseased vessels. The concns. of carotenoids and GSH-Px, as well as the .alpha./.**gamma**.-**tocopherol** ratio were also significantly assocd. Although conditional logistic regression anal. suggested low levels of HDL-cholesterol to be a significant coronary risk factor (OR=5.1, 95% CI=1.09-24.3), the TAS level showed no significant independent contribution to CAD. This study demonstrated an assocn. of antioxidant parameters with the atherosclerosis progression, however, it did not confirm antioxidants as an independent risk factor for CAD event.
CC 14 (Mammalian Pathological Biochemistry)
RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L46 ANSWER 2 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 2002:106843 HCAPLUS
TI Plasma status of retinol, .alpha.- and .**gamma**.-**tocopherols**, and main carotenoids to first myocardial infarction: case control and follow-up study
AU Ruiz Rejon, F.; Martin-Pena, G.; Granado, F.; Ruiz-Galiana, J.; Blanco, I.; Olmedilla, B.
CS Servicio de Cardiologia, Servicio de Medicina Interna, Seccion Nutricion, Hospital de Mostoles, Unidad de Vitaminas, Clinica Puerta de Hierro, Madrid, Spain
SO Nutrition (New York, NY, United States) (2002), 18(1), 26-31
CODEN: NUTRER; ISSN: 0899-9007
PB Elsevier Science Inc.
DT Journal
LA English

AB OBJECTIVE: Epidemiol. studies have suggested that dietary intake and plasma concns. of antioxidants have an inverse relation with coronary **heart disease**. To test whether fat-sol. antioxidants can play a role against the occurrence of myocardial infarction (MI), we measured plasma levels of retinol, tocopherols, and individual carotenoids in MI patients.METHODS: A case-control and follow-up study of patients in the Mostoles area (Madrid, Spain). One hundred six patients (62 after 1 y) and 104 control subjects participated in the study. Blood samples were collected after overnight fast or during the first 24 h of MI onset for biochem. profiles of retinol, .alpha.- and .**gamma**.-**tocopherols**, and carotenoid by means of a quality-controlled high-performance liq. chromatog.RESULTS: During the acute phase after MI onset, plasma levels of retinol, .**gamma**.-**tocopherol**, and xanthophylls (lutein/zeaxanthin and .beta.-cryptoxanthin) decreased, whereas .alpha.-tocopherol, .alpha.-carotene, .beta.-carotene, and lycopene showed levels similar to those of control subjects. Logistic regression anal. showed low concns. of .**gamma**.-**tocopherol** (and retinol) in plasma as the only statistically significant factor assocd. with MI, after adjusting for traditional risk factors. However, 1 y later, the MI patients showed a general improvement in plasma lipids and fat-sol. antioxidant status, and none of the analytes was assocd. with MI.CONCLUSIONS: The decreased plasma status of retinol, .**gamma**.-**tocopherol**, and xanthophylls during the acute phase of MI normalized the year after the MI event, suggesting that most subjects had followed an overall healthier lifestyle and dietary pattern. The results also raise concerns on the usefulness of these plasma compds. as specific, relevant, and predictive markers in relation to coronary **heart disease**.

CC 14 (Mammalian Pathological Biochemistry)

RE.CNT 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L46 ANSWER 3 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 2001:744482 HCAPLUS
DN 136:199429
TI Effects of dietary sesame seeds on plasma tocopherol levels
AU Cooney, Robert V.; Custer, Laurie J.; Okinaka, Leila; Franke, Adrian A.
CS University of Hawaii Cancer Research Center, Honolulu, HI, 96813, USA
SO Nutrition and Cancer (2001), 39(1), 66-71
CODEN: NUCADQ; ISSN: 0163-5581
PB Lawrence Erlbaum Associates, Inc.
DT Journal
LA English

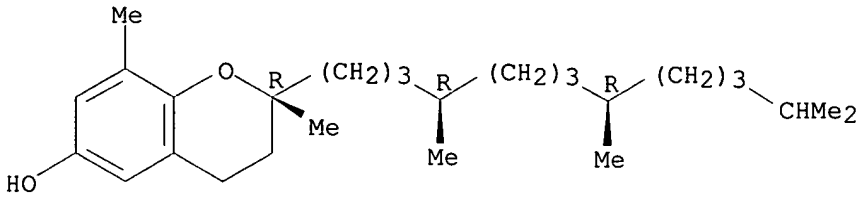
AB The tocopherols, major vitamers of vitamin E, may play a role in the prevention of human aging-related diseases such as cancer and **heart disease**, but little is known about determinants of their blood plasma concns. Animal studies suggest that dietary sources of .**gamma**.-**tocopherol** can affect plasma levels of this tocopherol and its vitamin E functional activity. To det. whether blood plasma levels of tocopherols in humans are similarly altered, 9 subjects (5 men, 4 women; 28-51 yr old) were given muffins contg. equiv. amts. of .**gamma**.-**tocopherol** from sesame seeds, walnuts, or soybean oil. Consumption of as little as 5 mg .**gamma**.-**tocopherol**/day over 3-day period from sesame seeds, but not from walnuts or soybean oil, elevated blood serum .**gamma**.-**tocopherol** levels by 19.1% and depressed plasma .**beta**.-**tocopherol** levels by 34%. No significant changes in baseline or post-intervention blood plasma levels of cholesterol, triglycerides, or carotenoids were seen in any intervention group. All subjects consuming

sesame seed-contg. muffins had detectable levels of the sesame lignan sesamolin in blood plasma. Thus, consumption of moderate amts. of sesame seeds may increase blood plasma **.gamma.-tocopherol** levels and alter plasma tocopherol ratios in humans. This is consistent with the effects of dietary sesame seeds obsd. in rats, leading to elevated blood plasma **.gamma.-tocopherol** levels and enhanced vitamin E biol. activity.

IT **119-13-1, .delta. Tocopherol 148-03-8, .beta. Tocopherol 7616-22-0, .gamma. Tocopherol**
RL: BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study); USES (Uses)
(dietary sesame seeds effects on blood plasma tocopherol levels in humans)

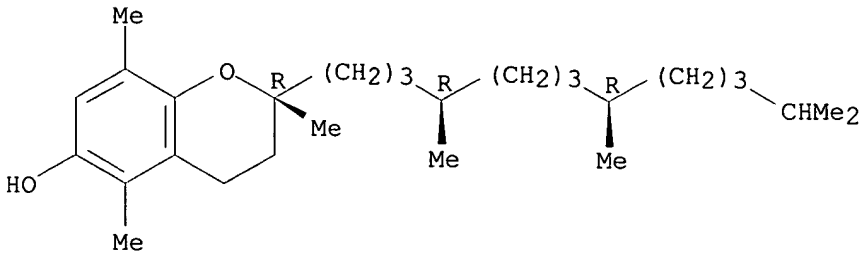
RN 119-13-1 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,8-dimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

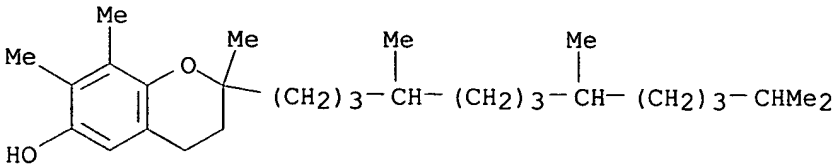


RN 148-03-8 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,5,8-trimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)-rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



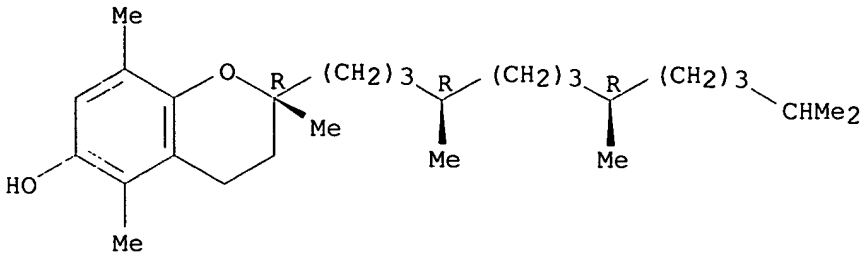
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 18-2 (Animal Nutrition)
IT 59-02-9, .alpha. Tocopherol **119-13-1, .delta. Tocopherol** **148-03-8, .beta. Tocopherol** 526-07-8, Sesamolin 607-80-7, Sesamin **7616-22-0, .gamma. Tocopherol**
RL: BSU (Biological study, unclassified); FFD (Food or feed use); BIOL (Biological study); USES (Uses)
(dietary sesame seeds effects on blood plasma tocopherol levels in humans)
RE.CNT 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L46 ANSWER 4 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 2000:763733 HCAPLUS
DN 134:51328
TI Antioxidants and herbal extracts protect HT-4 neuronal cells against glutamate-induced cytotoxicity
AU Kobayashi, Michael S.; Han, Derick; Packer, Lester
CS Membrane Bioenergetics Group, Department of Molecular and Cell Biology, University of California, Berkeley, CA, 94720-3200, USA
SO Free Radical Research (2000), 32(2), 115-124
CODEN: FRARER; ISSN: 1071-5762
PB Harwood Academic Publishers
DT Journal
LA English
AB Antioxidant therapy has been shown to be beneficial in neurol. disorders including Alzheimer's disease and **cerebral ischemia.** Glutamate-induced cytotoxicity in HT-4 neuronal cells has been previously demonstrated to be due to oxidative stress caused by depletion of cellular glutathione (GSH). The present study demonstrates that a wide variety of antioxidants inhibit glutamate-induced cytotoxicity in HT-4 neuronal cells. Low concns. of .alpha.-tocopherol and its analogs were highly effective in protecting neuronal cells against cytotoxicity. Purified flavonoids and herbal exts. of Gingko biloba (EGb 761) and French maritime pine bark (Pycnogenol) were also effective. We have previously shown that pro-glutathione agents can spare GSH and protect cells from glutamate insult in a C6 glial cell model. The protective effects of nonthiol-based antioxidants tested in the HT-4 line were not mediated via GSH level modulation. In contrast, protective effects of thiol-based pro-glutathione agents .alpha.-lipoic acid (LA) and N-acetyl cysteine (NAC) corresponded with a sparing effect on GSH levels in glutamate-treated HT-4 cells. Glutamate-induced cytotoxicity in HT-4 cells is a useful model system for testing compds. or mixts. for antioxidant activity.
IT **148-03-8, .beta.-Tocopherol**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(antioxidants and herbal exts. protect HT-4 neuronal cells against glutamate-induced cytotoxicity)
RN 148-03-8 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,5,8-trimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)-rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



CC 1-11 (Pharmacology)
IT 50-81-7, L-Ascorbic acid, biological studies 59-02-9, .alpha.-Tocopherol 70-51-9, Desferrioxamine 117-39-5, Quercetin 128-37-0, BHT, biological studies **148-03-8, .beta.-Tocopherol** 153-18-4, Rutin 616-91-1, N-Acetyl cysteine 1200-22-2, .alpha.-Lipoic acid 122933-57-7, EGb 761 152905-68-5, PMC 174882-69-0, Pycnogenol
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(antioxidants and herbal exts. protect HT-4 neuronal cells against glutamate-induced cytotoxicity)
RE.CNT 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

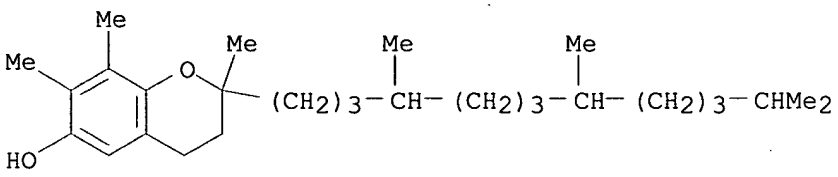
L46 ANSWER 5 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 2000:759916 HCAPLUS
DN 134:36796
TI **.gamma.-Tocopherol** and its major metabolite, in contrast to .alpha.-tocopherol, inhibit cyclooxygenase activity in macrophages and epithelial cells
AU Jiang, Qing; Elson-Schwab, Ilan; Courtemanche, Chantal; Ames, Bruce N.
CS Division of Biochemistry and Molecular Biology, University of California, Berkeley, CA, 94720, USA
SO Proceedings of the National Academy of Sciences of the United States of America (2000), 97(21), 11494-11499
CODEN: PNASA6; ISSN: 0027-8424
PB National Academy of Sciences
DT Journal
LA English
AB Cyclooxygenease-2 (COX-2)-catalyzed synthesis of prostaglandin E2 (PGE2) plays a key role in inflammation and its assocd. diseases, such as cancer and vascular **heart disease.** Here we report that .**gamma.-tocopherol** (.gamma.T) reduced PGE2 synthesis in both lipopolysaccharide (LPS)-stimulated RAW264.7 macrophages and IL-1.beta.-treated A549 human epithelial cells with an apparent IC50 of 7.5 and 4 .mu.M, resp. The major metabolite of dietary .gamma.T, 2,7,8-trimethyl-2-(.beta.-carboxyethyl)-6-hydroxychroman (.**gamma.-CEHC**), also exhibited an inhibitory effect, with an IC50 of .apprxeq.30 .mu.M in these cells. In contrast, .alpha.-tocopherol at 50 .mu.M slightly reduced (25%) PGE2 formation in macrophages, but had no effect in epithelial cells. The inhibitory effects of .gamma.T and .**gamma.-CEHC** stemmed from their inhibition of COX-2 activity, rather than affecting protein expression or substrate availability, and appeared to be independent of antioxidant activity. .**gamma.-CEHC** also inhibited PGE2 synthesis when exposed for 1 h to COX-2-preinduced cells followed by the addn. of arachidonic acid (AA), whereas under similar conditions, .gamma.T required an 8- to 24-h incubation period to cause the inhibition. The inhibitory potency of .gamma.T and .**gamma.-CEHC** was diminished by an

increase in AA concn., suggesting that they might compete with AA at the active site of COX-2. We also obsd. a moderate redn. of nitrite accumulation and suppression of inducible nitric oxide synthase expression by .gamma.T in lipopolysaccharide-treated macrophages. These findings indicate that .gamma.T and its major metabolite possess anti-inflammatory activity and that .gamma.T at physiol. concns. may be important in human disease prevention.

IT **7616-22-0, .gamma.-Tocopherol**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**.gamma.-Tocopherol** and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

RN 7616-22-0 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 1-7 (Pharmacology)

ST **gamma tocopherol** cyclooxygenase 2 antiinflammatory

IT Antioxidants
(pharmaceutical; **.gamma.-Tocopherol** and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 39391-18-9
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(cyclooxygenase-2; **.gamma.-Tocopherol** and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 59-02-9, .alpha.-Tocopherol
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**.gamma.-Tocopherol** and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 178167-88-9
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); MFM (Metabolic formation); THU (Therapeutic use); BIOL (Biological study); FORM (Formation, nonpreparative); USES (Uses)
(**.gamma.-Tocopherol** and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT **7616-22-0, .gamma.-Tocopherol**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**.gamma.-Tocopherol** and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)

IT 363-24-6, PGE2 41598-07-6, PGD2 125978-95-2, Nitric oxide synthase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**.gamma.-Tocopherol** and its major metabolite

inhibit cyclooxygenase activity in macrophages and epithelial cells)
IT 155976-51-5, 8-Isoprostane
RL: BSU (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative)
(.gamma.-**Tocopherol** and its major metabolite inhibit cyclooxygenase activity in macrophages and epithelial cells)
RE.CNT 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L46 ANSWER 6 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 2000:604734 HCAPLUS
DN 133:321303
TI Effect of vitamin E on the development of atherosclerosis
AU Ozer, Nesrin Kartal; Azzi, Angelo
CS Faculty of Medicine, Department of Biochemistry, Marmara University, Haydarpasa, Istanbul, 81326, Turk.
SO Toxicology (2000), 148(2-3), 179-185
CODEN: TXCYAC; ISSN: 0300-483X
PB Elsevier Science Ireland Ltd.
DT Journal
LA English
AB The development of atherosclerosis is a multifactorial process in which both elevated blood plasma cholesterol levels and proliferation of vascular smooth muscle cells play a central role. Numerous studies have suggested the involvement of oxidative processes in the pathogenesis of atherosclerosis and esp. of oxidized low-d. lipoproteins. Some epidemiol. studies have shown an assocn. between high dietary intake or high blood serum concns. of vitamin E and lower rates of ischemic **heart disease**. Strong protective effects of high vitamin E doses against the risk of fatal and nonfatal myocardial infarction have been reported. In this study, the incubation of vascular smooth muscle cells in the presence of .alpha.-tocopherol resulted in inhibition of cell proliferation and protein kinase C activity. Since .**beta.-tocopherol** and probucol were not inhibitory, the effects of .alpha.-tocopherol were likely due to a non-oxidant mechanism. To understand the protective role of .alpha.-tocopherol against atherosclerosis in vivo, studies in rabbits were carried out. Atherosclerosis was induced by a low-vitamin E diet contg. 2% cholesterol. Three other groups were fed diets with 2% cholesterol combined with 50 mg vitamin E (Ephynal)/kg i.m., 1% probucol in feed, or 50 mg vitamin E/kg plus 1% probucol. After 4 wk, the aortas were analyzed by microscopy for atherosclerotic lesions. Samples of the aortic media were analyzed for protein kinase C activity. The aortas of cholesterol-fed rabbits had typical atherosclerotic lesions detected by microscopic examn. and the medial smooth muscle cells had increased protein kinase C activity. Vitamin E fully prevented the cholesterol induced atherosclerotic lesions and the induction of protein kinase C activity, while probucol was not effective. Thus, the protective effects of vitamin E against hypercholesterolemic atherosclerosis is not due to another antioxidant (such as probucol) and may not be linked to the antioxidant properties of vitamin E. The effects obsd. at the level of smooth muscle cells in vitro and ex-vivo suggest an involvement of signal transduction events in the protective effects of vitamin E against atherosclerosis.
CC 18-2 (Animal Nutrition)
Section cross-reference(s): 14
RE.CNT 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

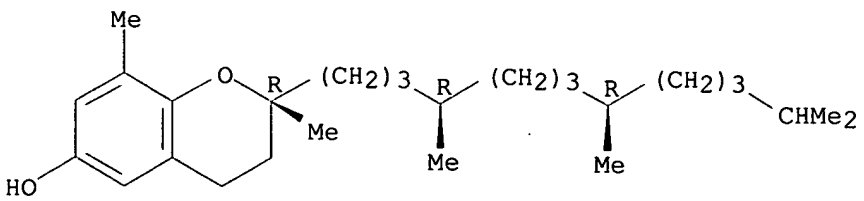
L46 ANSWER 7 OF 32 HCAPLUS COPYRIGHT 2002 ACS

AN 2000:238052 HCAPLUS
DN 132:260686
TI Use of **.gamma.-tocopherol** and its oxidative metabolite LLU-.alpha. in the treatment of natriuretic disease
IN Wechter, William J.
PA Loma Linda University Medical Center, USA
SO U.S., 21 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 6048891	A	20000411	US 1998-215608	19981217
	US 6242479	B1	20010605	US 1999-461645	19991214
	WO 2000035444	A1	20000622	WO 1999-US30100	19991216
	W: AU, CA, JP				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	EP 1140065	A1	20011010	EP 1999-968905	19991216
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	US 2001031782	A1	20011018	US 2001-814330	20010321
PRAI	US 1998-215608	A1	19981217		
	US 1999-461645	A1	19991214		
	WO 1999-US30100	W	19991216		

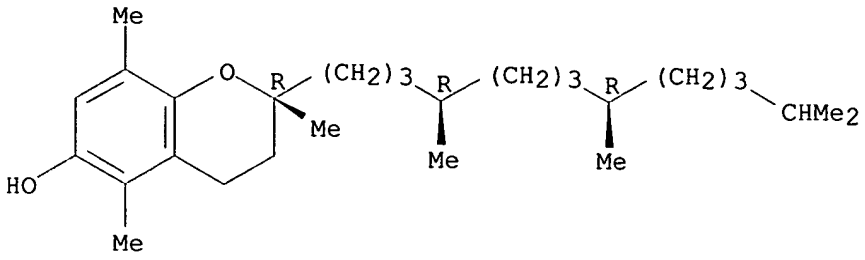
OS MARPAT 132:260686
AB The invention is generally related to the discovery of the therapeutic benefit of administering **.gamma.-tocopherol** and **.gamma.-tocopherol** derivs. More specifically, the use of **.gamma.-tocopherol** and racemic LLU-.alpha., (S)-LLU-.alpha., or **.gamma.-tocopherol** derivs. as antioxidants and nitrogen oxide scavengers which treat and prevent high blood pressure, **thromboembolic** disease, cardiovascular disease, cancer, natriuretic disease, the formation of neuropathol. lesions, and a reduced immune system response are disclosed.
IT **119-13-1**, **.delta.-Tocopherol 148-03-8**, **.beta.-Tocopherol 7616-22-0**, **.gamma.-Tocopherol**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**.gamma.-tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)
RN 119-13-1 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,8-dimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

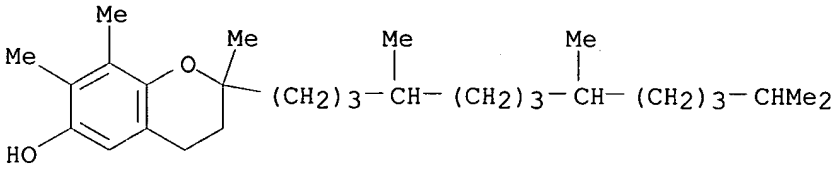


RN 148-03-8 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,5,8-trimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)-rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



IC ICM A61K031-35
ICS A61K031-355
NCL 514456000
CC 1-8 (Pharmacology)
Section cross-reference(s): 27, 63
ST **gamma tocopherol** metabolite LLUalpha natriuretic disease; antioxidant NO scavenger tocopherol metabolite LLUalpha; hypotensive **thromboembolic** disease tocopherol metabolite LLUalpha; cardiovascular disease cancer tocopherol metabolite LLUalpha; neuropathol lesion immunomodulation tocopherol metabolite LLUalpha
IT **Heart, disease**
(angina pectoris; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)
IT Resolution (separation)
(chromatog.; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)
IT **Heart, disease**
(failure; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)
IT Kidney, disease
(glomerulus, ineffective glomerular filtration; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)
IT Kidney, disease
(ineffective renal perfusion; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)
IT Urine
(natriuretic compds. isolation from; .**gamma**.-

tocopherol and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT Diuretics
(natriuretics; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT Kidney, disease
(nephrotic syndrome; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT Drug delivery systems
(oral; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT Drug delivery systems
(parenterals; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT Anti-ischemic agents
Antihypertensives
Cirrhosis
(.**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT Tocopherols
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(.**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT 4072-32-6P 178167-78-7P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(prepn. and reaction; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT 108-24-7, Acetic anhydride 526-75-0, 2,3-Dimethylphenol 608-43-5 697-82-5, 2,3,5-Trimethylphenol 700-13-0, 2,3,5-Trimethyl-1,4-hydroquinone 1073-11-6 178167-90-3
RL: RCT (Reactant); RACT (Reactant or reagent)
(reaction; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT 9000-83-3, ATPase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(sodium/potassium; .**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT 178167-75-4P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PUR (Purification or recovery); RCT (Reactant); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); RACT (Reactant or reagent); USES (Uses)
(.**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT 178167-88-9P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); PUR (Purification or recovery); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(.**gamma**.-**tocopherol** and oxidative metabolite LLU-.alpha. in treatment of natriuretic disease)

IT 59-02-9, .alpha.-Tocopherol **119-13-1**, .**delta**.-**Tocopherol** **148-03-8**, .**beta**.-**Tocopherol** **7616-22-0**, .**gamma**.-**Tocopherol**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES

(Uses)
(.**gamma**.-**tocopherol** and oxidative metabolite
LLU-.alpha. in treatment of natriuretic disease)
IT 7440-23-5, Sodium, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(.**gamma**.-**tocopherol** and oxidative metabolite
LLU-.alpha. in treatment of natriuretic disease)
IT 170427-68-6P, Natriuretic peptide LLU-.gamma.
RL: PUR (Purification or recovery); PREP (Preparation)
(.**gamma**.-**tocopherol** and oxidative metabolite
LLU-.alpha. in treatment of natriuretic disease)
IT 178167-79-8P 178167-89-0P
RL: PUR (Purification or recovery); SPN (Synthetic preparation); PREP (Preparation)
(.**gamma**.-**tocopherol** and oxidative metabolite
LLU-.alpha. in treatment of natriuretic disease)
IT 22625-17-8P 178167-76-5P 178167-77-6P 178167-80-1P 178232-68-3P
RL: SPN (Synthetic preparation); PREP (Preparation)
(.**gamma**.-**tocopherol** and oxidative metabolite
LLU-.alpha. in treatment of natriuretic disease)
RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

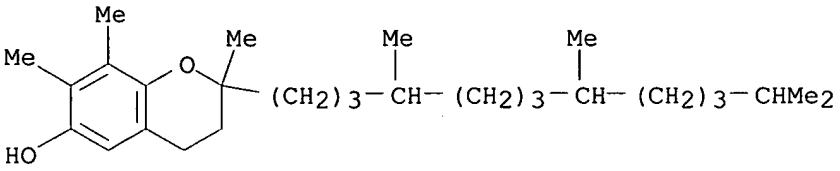
L46 ANSWER 8 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 2000:221501 HCAPLUS
DN 133:38178
TI Modulation by .alpha.- and .**gamma**.-**tocopherol** and oxidized low-density lipoprotein of apoptotic signaling in human coronary smooth muscle cells
AU de Nigris, F.; Franconi, F.; Maida, I.; Palumbo, G.; Anania, V.; Napoli, C.
CS Department of Clinical and Experimental Medicine, University of Naples, Naples, Italy
SO Biochemical Pharmacology (2000), 59(11), 1477-1487
CODEN: BCPCA6; ISSN: 0006-2952
PB Elsevier Science Inc.
DT Journal
LA English
AB Apoptosis may play an important role in atherogenesis. Oxidized low-d. lipoprotein (oxLDL) promotes apoptosis in the arterial wall in addn. to several other proatherogenic effects. Tocopherol supplements have been suggested to protect against coronary **heart disease** (CHD) in epidemiol. studies. The effects of oxLDL and .alpha.- and .**gamma**.-**tocopherol** on apoptotic signaling pathways are poorly understood. Thus, the goal of the study was to investigate these pathways in the presence of copper-oxidized LDL and tocopherols in human coronary smooth muscle cells (SMC). We showed that oxLDL-mediated apoptosis, assessed by DNA fragmentation, terminal deoxynucleotidyl transferase (TdT)-mediated dUTP nick end labeling (TUNEL) assay, and caspase activation stimulated several transcription factors and proapoptotic dynamic movements of the Bcl-2 family proteins through the mitogen-activated protein kinase (MAPK) and Jun kinase pathways. .alpha.-Tocopherol and .**gamma**.-**tocopherol** significantly reduced these mol. events and cell death effectors caspase-3 and -8. Under our exptl. conditions, .alpha.-tocopherol was significantly more effective than .**gamma**.-**tocopherol**, and oxLDL-mediated apoptosis increased c-Jun, cAMP-responsive element-binding, Ets-like element kinase-dependent 7, and activating transcription factor-2

proteins as well as nuclear activity of the activated protein-1 complex in human coronary SMC. Moreover, our results demonstrate that tocopherols may exert their antiatherogenic effects at least in part via redn. of the MAPK and JunK cascade together with a protective profile of apoptotic genes of the Bcl-2 family. These data are consistent with the beneficial effects of tocopherols on atherogenesis seen in exptl. studies and on CHD in epidemiol. surveys.

IT **7616-22-0, .gamma.-Tocopherol**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(modulation by .alpha.- and **.gamma.-tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)

RN 7616-22-0 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 1-12 (Pharmacology)

IT Transcription factors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(AP-1 (activator protein 1); modulation by .alpha.- and **.gamma.-tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)

IT Transcription factors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(ATF-2 (activating transcription factor 2); modulation by .alpha.- and **.gamma.-tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)

IT Transcription factors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(NF-.kappa.B (nuclear factor .kappa.B); modulation by .alpha.- and **.gamma.-tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)

IT Antiarteriosclerotics
(antiatherosclerotics; modulation by .alpha.- and **.gamma.-tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)

IT Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(bcl-2; modulation by .alpha.- and **.gamma.-tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)

IT Artery, disease
(coronary; modulation by .alpha.- and **.gamma.-tocopherol** and oxidized low-d. lipoprotein of apoptotic

signaling in human coronary smooth muscle cells)
IT Transcription factors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(gene elk-1; modulation by .alpha.- and .**gamma**.-**tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)
IT Lipoproteins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(low-d., oxidized; modulation by .alpha.- and .**gamma**.-**tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)
IT Apoptosis
Signal transduction, biological
(modulation by .alpha.- and .**gamma**.-**tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)
IT Antioxidants
(pharmaceutical; modulation by .alpha.- and .**gamma**.-**tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)
IT 59-02-9, .alpha.-Tocopherol **7616-22-0**, .**gamma**.-**Tocopherol**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(modulation by .alpha.- and .**gamma**.-**tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)
IT 142243-02-5, Mitogen-activated protein kinase 155215-87-5 169592-56-7, Caspase-3 179241-78-2, Caspase-8
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(modulation by .alpha.- and .**gamma**.-**tocopherol** and oxidized low-d. lipoprotein of apoptotic signaling in human coronary smooth muscle cells)
RE.CNT 61 THERE ARE 61 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L46 ANSWER 9 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 2000:219062 HCAPLUS
DN 132:256003
TI Water-soluble compositions of bioactive lipophilic compounds
IN Borowy-Borowski, Henryk; Sikorska-Walker, Marianna; Walker, P. Roy
PA National Research Council of Canada, Can.
SO U.S., 19 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 6045826	A	20000404	US 1999-285244	19990402
	WO 2000061189	A2	20001019	WO 2000-CA76	20000203
	WO 2000061189	A3	20010111		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA,

MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1159006	A2	20011205	EP 2000-901445	20000203	
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
BR 2000009532	A	20011226	BR 2000-9532	20000203	
US 6191172	B1	20010220	US 2000-511239	20000223	
FI 2001001914	A	20011119	FI 2001-1914	20010928	
NO 2001004780	A	20011127	NO 2001-4780	20011001	
PRAI US 1999-285244	A	19990402			
WO 2000-CA76	W	20000203			

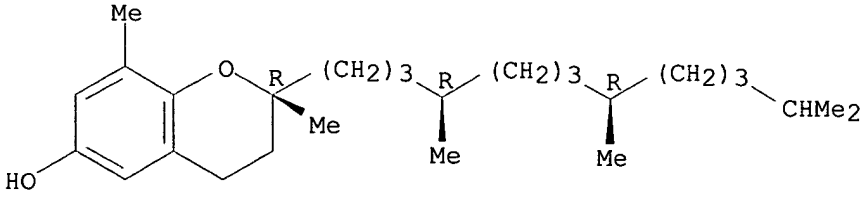
OS MARPAT 132:256003

AB Water-sol. compns. comprising a lipophilic compd. and a solubilizing agent of the general formula: {X-OOC-[(CH2)n-COO]m}p-Y wherein: X is a residue of a hydrophobic moiety, Y is a residue of a hydrophilic moiety, p is 1 or 2, m is 0 or 1, and n is an integer greater than or equal to 0 are disclosed. The lipophilic compd. is preferably selected from the group consisting of water-insol. ubiquinones, ubiquinols, vitamins, provitamins, polyene macrolide antibiotics, and mixts. thereof. The hydrophobic moiety is preferably a sterol or a tocopherol and the hydrophilic moiety is preferably a polyalkylene glycol. In preferred embodiments, the sterol is cholesterol or sitosterol, the tocopherol is a-(+)-tocopherol, the polyalkylene glycol is a polyethylene glycol or its Me monoether having an av. mol. wt. between 600 and 1000, p is equal to 1 or 2, m is equal to 0 or 1 and n is an integer between 2 and 18. A water sol. compn. contained vitamin E 0.10, polyoxyethyanyl-.alpha.-tocopheryl sebacate (prepn. given) 0.60, vitamin E 0.22, polyoxyetahanyl-.alpha.-tocopheryl sebacate 1.00 g, THF 2.50, and water 35.00 mL.

IT **119-13-1, .delta.-Tocopherol 148-03-8, .beta.-Tocopherol 7616-22-0, .gamma.-Tocopherol**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(water-sol. compns. of bioactive lipophilic compds.)

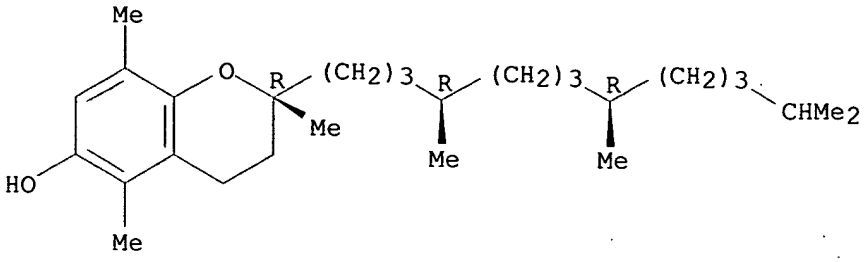
RN 119-13-1 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,8-dimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

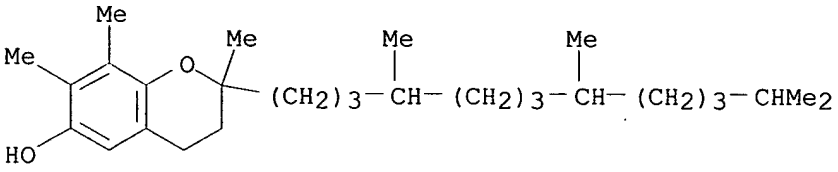


RN 148-03-8 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,5,8-trimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)-rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



IC ICM A61K009-48
ICS A61K009-14
NCL 424451000
CC 63-6 (Pharmaceuticals)
IT Drug bioavailability
Dyes
Flavor
Heart, disease
Hypercholesterolemia
Infection
Lubricants
Neoplasm
Preservatives
Sweetening agents
(water-sol. compns. of bioactive lipophilic compds.)
IT 50-81-7, L-Ascorbic acid, biological studies 57-87-4, Ergosterol 57-88-5, Cholesterol, biological studies 59-02-9, .alpha.-(+)-Tocopherol 83-48-7, Stigmasterol **119-13-1, .delta.-Tocopherol** **148-03-8, .beta.-Tocopherol** 303-98-0, Coenzyme q 10 434-16-2, 7-Dehydrocholesterol 474-62-4, Campesterol 1397-89-3, Amphotericin b 1400-61-9, Nystatin 1403-17-4, Candicidin 1406-16-2, Vitamin d 1406-18-4, Vitamin e 2074-53-5, dl-.alpha.-Tocopherol **7616-22-0, .gamma.-Tocopherol** 9004-74-4 11103-57-4, Provitamin a 12001-79-5, Vitamin k 106602-88-4 146846-92-6 263015-34-5 263015-35-6 263015-36-7 263015-37-8 263015-38-9
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(water-sol. compns. of bioactive lipophilic compds.)
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L46 ANSWER 10 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 2000:134517 HCAPLUS
DN 132:148749
TI Fluorometric determination of lipid oxidizability in biological systems

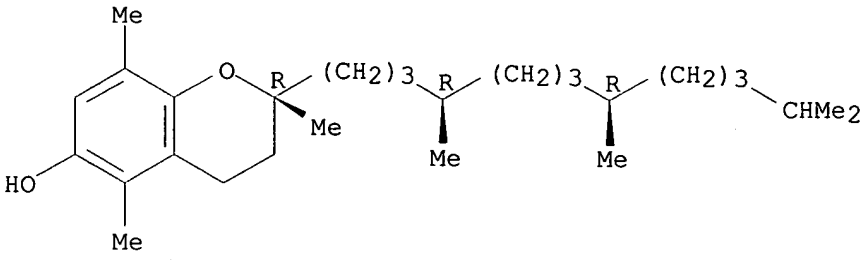
using diphenylhexatriene
IN Hermetter, Albin; Hofer, Gerald; Lichtenberg, Dov
PA Austria
SO Austrian, 10 pp.
CODEN: AUXXAK
DT Patent
LA German
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI AT 405693	B	19991025	AT 1994-1875	19941004
AT 9401875	A	19990215		

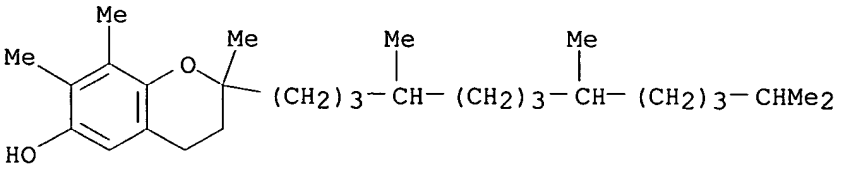
AB The invention concerns the detn. of lipid oxidizability in biol. systems, e.g. in lipoproteins, by using diphenylhexatriene and its lipid-derivs. as markers for detecting the progress of oxidn. via the decreasing fluorescent signal. The method is used for cells, serum, and food samples for measuring the effects of oxidants or antioxidants.

IT **148-03-8, .beta.-Tocopherol 7616-22-0 , .gamma.-Tocopherol**
RL: BSU (Biological study, unclassified); BIOL (Biological study) (fluorometric detn. of lipid oxidizability in biol. systems using diphenylhexatriene)
RN 148-03-8 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,5,8-trimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)-rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



IC ICM G01N033-92
CC 9-5 (Biochemical Methods)
Section cross-reference(s): 1, 6, 14, 17
IT Alzheimer's disease
Animal tissue
Antioxidants
Atherosclerosis

Blood
Blood plasma
Blood serum
Cell
Diagnosis
Fluorometry
Food analysis
Heart, disease
Neoplasm
Oxidizability
Oxidizing agents
Sickle cell anemia
(fluorometric detn. of lipid oxidizability in biol. systems using diphenylhexatriene)

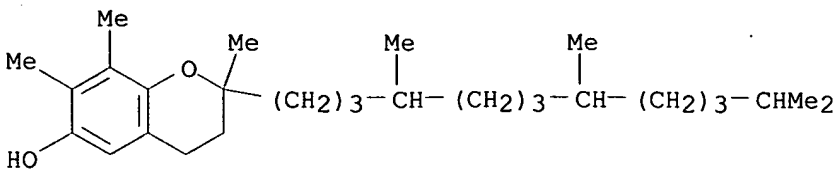
IT 50-53-3, Chlorpromazine, biological studies 52-90-4, L-Cysteine, biological studies 57-13-6, Urea, biological studies 59-02-9, .alpha.-Tocopherol 60-87-7, Promethazine 91-53-2, Ethoxyquin **148-03-8, .beta.-Tocopherol 7616-22-0, .gamma.-Tocopherol** 9001-05-2, Catalase 9003-99-0, Peroxidase 9054-89-1, Superoxide dismutase 25013-16-5 258280-06-7
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(fluorometric detn. of lipid oxidizability in biol. systems using diphenylhexatriene)

L46 ANSWER 11 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1999:592299 HCAPLUS
DN 131:285861
TI Reversals of age-related declines in neuronal signal transduction, cognitive, and motor behavioral deficits with blueberry, spinach, or strawberry dietary supplementation
AU Joseph, James A.; Shukitt-Hale, Barbara; Denisova, Natalia A.; Bielinski, Donna; Martin, Antonio; McEwen, John J.; Bickford, Paula C.
CS USDA - Human Nutrition Research Center on Aging, Tufts University, Boston, MA, 02111, USA
SO Journal of Neuroscience (1999), 19(18), 8114-8121
CODEN: JNRSDS; ISSN: 0270-6474
PB Society for Neuroscience
DT Journal
LA English
AB Age-related neuronal and behavioral decrements result from oxidative stress that may be ameliorated by antioxidants. Rats fed fruit and vegetable exts. with high antioxidant activity for 8 mo beginning at 6 mo of age have delayed age-related declines in the neuronal and cognitive functions. Strawberry, spinach, and blueberry supplements fed at 14.8, 9.1, or 18.6 g dried aq. ext./kg feed, resp. were fed for 8 wk to 19-mo-old Fischer 344 rats. The exts. were also effective in reversing age-related deficits in several neuronal and behavioral parameters, including oxotremorine enhancement of K+-evoked release of dopamine from brain striatal slices, carbachol-stimulated GTPase activity, striatal 45Ca buffering in striatal synaptosomes, motor behavioral performance on the rod walking and accelerod tasks, and Morris water maze behavior performance. Thus, in addn. to their known beneficial effects on cancer and **heart disease**, phytochems. present in antioxidant-rich foods may be beneficial in reversing the course of neuronal and behavioral aging.
IT **7616-22-0**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(dietary blueberry, spinach and strawberry exts. supplements reverse age-related declines in neuronal signal transduction, cognition and motor behavior)

RN 7616-22-0 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 18-7 (Animal Nutrition)

IT 51-61-6, Dopamine, biological studies 59-02-9, .alpha. Tocopherol 70-18-8, Gsh, biological studies 7440-70-2, Calcium, biological studies **7616-22-0** 9059-32-9, Gtpase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(dietary blueberry, spinach and strawberry exts. supplements reverse age-related declines in neuronal signal transduction, cognition and motor behavior)

RE.CNT 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L46 ANSWER 12 OF 32 HCAPLUS COPYRIGHT 2002 ACS

AN 1999:484621 HCAPLUS

DN 131:115727

TI Effects of a soy milk supplement on plasma cholesterol levels and oxidative DNA damage in men. A pilot study

AU Mitchell, J. H.; Collins, A. R.

CS Rowett Research Institute, Bucksburn, AB21 9SB, UK

SO European Journal of Nutrition (1999), 38(3), 143-148
CODEN: EJNUFZ; ISSN: 1436-6207

PB Dr. Dietrich Steinkopff Verlag GmbH & Co. KG

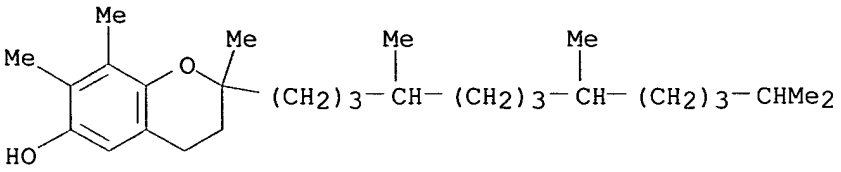
DT Journal

LA English

AB Phytoestrogens are a major component of Asian diets and may be protective against certain hormone-dependent cancers (breast and prostate) and coronary **heart disease**. They may also have antioxidant function in scavenging potentially harmful free radicals and thus decreasing oxidative attack on DNA. A pilot study to det. the effects of a phytoestrogen supplement, in the form of soy milk, on plasma LDL and HDL cholesterol levels and DNA damage in men. Healthy men participated in the study and were assigned to one of 3 groups consuming 1 L of either soy milk, rice dream (vegetable protein control), or semi-skimmed cow's milk (animal protein control) each day for 4 wk. The soy supplement caused increases in blood plasma genistein and daidzein despite considerable inter-individual variation. Supplementation with soy resulted in a decrease in oxidative damage to DNA bases detected using the comet assay compared with controls. There was no effect of the soy supplement on plasma cholesterol or triglycerides in comparison with control groups. Thus, a 4 wk soy milk supplementation in healthy volunteers does not alter serum cholesterol levels but can have a protective effect against oxidative DNA damage in lymphocytes.

IT **7616-22-0, .gamma.-Tocopherol**

RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(soy milk effect on blood antioxidants)
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 18-7 (Animal Nutrition)
IT 59-02-9, .alpha.-Tocopherol **7616-22-0**, **.gamma.-Tocopherol**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(soy milk effect on blood antioxidants)
RE.CNT 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

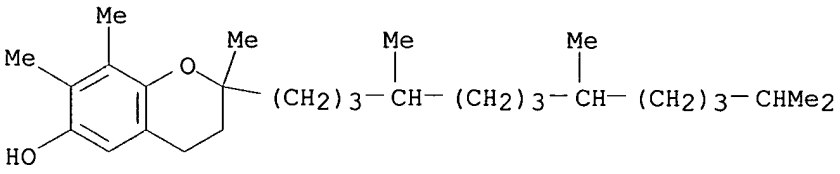
L46 ANSWER 13 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1999:483847 HCAPLUS
DN 131:285838
TI Gender differences in response to a hypercholesterolemic diet in hamsters: effects on plasma lipoprotein cholesterol concentrations and early aortic atherosclerosis
AU Wilson, Thomas A.; Nicolosi, Robert J.; Lawton, Carl W.; Babiak, John
CS Center for Chronic Disease Control, Department of Health and Clinical Science, University of Massachusetts, Lowell, MA, 01854, USA
SO Atherosclerosis (Shannon, Ireland) (1999), 146(1), 83-91
CODEN: ATHSBL; ISSN: 0021-9150
PB Elsevier Science Ireland Ltd.
DT Journal
LA English
AB Gender is a strong predictor of coronary **heart disease** (CHD) susceptibility and men are more likely to develop CHD compared to age-matched premenopausal women. To test whether similar gender differences exist in hamsters, 16 male and 16 female F1B 10-wk-old Golden Syrian hamsters were fed a hypercholesterolemic diet (HCD) contg. 10% coconut oil and 0.05% cholesterol for 12 wk. Blood plasma lipid and lipoprotein cholesterol concns., LDL oxidn. susceptibility, LDL tocopherol concns., LDL fatty acid compn., LDL particle size, plasma estradiol and testosterone concns., and early aortic atherosclerosis were analyzed. Female hamsters had lower plasma total cholesterol (TC) and non-HDL-cholesterol and greater HDL-cholesterol concns. compared to male hamsters (-15, -33, and +33%; resp.). Female hamsters had greater LDL particles (4%), LDL C22:6 (21%) fatty acid content, and rate of LDL oxidn. (34%) compared to male hamsters. Female hamsters had higher concns. of plasma estradiol (49%) compared to male hamsters. Female hamsters also had less early aortic atherosclerosis compared to male hamsters (-77%). In female hamsters the aortic fatty streak formation was assocd. with plasma non-HDL-cholesterol levels (r=0.76), LDL particle size (r=-0.66), plasma TC levels (r=0.68), and lag phase of LDL oxidn. (r=0.84). In male hamsters the aortic fatty streak formation was assocd. with plasma

non-HDL-cholesterol levels (r=0.52), plasma TC (r=0.55), plasma glyceride levels (r=0.79), and LDL C22:6 (r=-0.78), with no assocn. with any measures of LDL oxidn. susceptibility. Thus, female hamsters have an improved plasma lipoprotein cholesterol profile, larger LDL particle size, and less early aortic atherosclerosis compared to male hamsters fed the same HCD.

IT **7616-22-0**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(dietary cholesterol effects on blood lipids and lipoproteins and gender differences in atherogenesis in hamsters)

RN 7616-22-0 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 18-5 (Animal Nutrition)
Section cross-reference(s): 14

IT 50-28-2, Estradiol, biological studies 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 58-22-0, Testosterone 59-02-9, .alpha. Tocopherol 544-63-8, Tetradecanoic acid, biological studies **7616-22-0** 27104-13-8 27213-43-0 28039-99-8 28984-77-2 31152-45-1 32839-18-2 32839-30-8
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(dietary cholesterol effects on blood lipids and lipoproteins and gender differences in atherogenesis in hamsters)

RE.CNT 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L46 ANSWER 14 OF 32 HCAPLUS COPYRIGHT 2002 ACS

AN 1999:440907 HCAPLUS

DN 131:198917

TI Vitamin E reduces plasma low density lipoprotein cholesterol, LDL oxidation, and early aortic atherosclerosis compared with black tea in hypercholesterolemic hamsters

AU Nicolosi, Robert J.; Lawton, Carl W.; Wilson, Thomas A.

CS Center for Chronic Disease Control, Department of Health and Clinical Science, University of Massachusetts, Lowell, MA, 01854, USA

SO Nutrition Research (New York) (1999), 19(8), 1201-1214
CODEN: NTRSDC; ISSN: 0271-5317

PB Elsevier Science Inc.

DT Journal

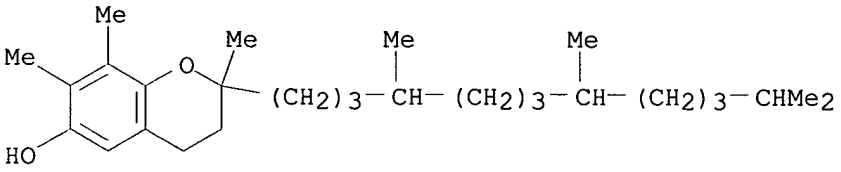
LA English

AB Dietary intake of tea polyphenols is inversely assocd. with the development of coronary **heart disease** via decreased low-d. lipoprotein (LDL) oxidn. Eighty male F1B Golden Syrian hamsters, 7 wk of age, were divided into 4 groups. The groups were fed semipurified hypercholesterolemic diets contg. 12% coconut oil, 3% sunflower oil, and 0.2% cholesterol (control), control + 0.625% wt./wt. brewed black tea (low

tea, control + 1.25% brewed black tea (high tea), or control + 0.044% .alpha.-tocopherol acetate (Vitamin E) for 10 wk. Hamsters fed the Vitamin E diet had decreased blood plasma LDL-cholesterol concns. by 18, 17, and 24% compared to the control, low tea, and high tea diet groups, resp. The aortic fatty streak area in the Vitamin E diet group was decreased by 36 and 45% compared to the control and low tea groups, resp. The lag phase of conjugated diene prodn. in the Vitamin E group was 41, 40, and 39% longer compared to the control, low tea, and high groups, resp. The rate of conjugated diene prodn. in the Vitamin E group was decreased by 63, 57, and 59% compared to the control, low tea, and high tea groups, resp. The max. amt. of conjugated dienes produced in the Vitamin E group was 14 and 22% lower compared to the control and low tea groups, resp. The Vitamin E group had 69, 71, and 65% greater concns. of LDL .alpha.-tocopherol compared to the control, low tea, and high tea groups, resp. Thus, dietary vitamin E supplementation decreased blood plasma LDL cholesterol concns., LDL oxidn., and early atherosclerosis compared to black tea consumption in the hypercholesterolemic hamster model. The antioxidant actions of vitamin E may be mediated by incorporation of vitamin E into LDL.

IT **7616-22-0**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(dietary vitamin E and black tea effects on blood plasma LDL cholesterol and LDL oxidn. and early aortic atherosclerosis in hypercholesterolemic hamsters)

RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 18-2 (Animal Nutrition)
Section cross-reference(s): 14

IT **7616-22-0**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(dietary vitamin E and black tea effects on blood plasma LDL cholesterol and LDL oxidn. and early aortic atherosclerosis in hypercholesterolemic hamsters)

RE.CNT 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

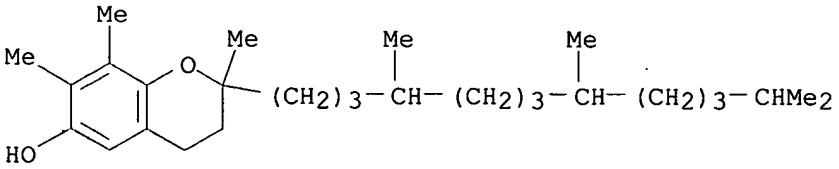
L46 ANSWER 15 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1999:423634 HCAPLUS
DN 131:168604
TI Lipophilic antioxidants in blood plasma as markers of atherosclerosis: the role of .alpha.-carotene and **.gamma.-tocopherol**
AU Kontush, Anatol; Spranger, Torsten; Reich, Axel; Baum, Katja; Beisiegel, Ulrike
CS Biochemisches Labor, Medizinische Kern- und Poliklinik, Universitatskrankenhaus Eppendorf, Hamburg, D-20246, Germany
SO Atherosclerosis (Shannon, Ireland) (1999), 144(1), 117-122

CODEN: ATHSBL; ISSN: 0021-9150
PB Elsevier Science Ireland Ltd.
DT Journal
LA English

AB Oxidative theory of atherosclerosis implies that plasma levels of lipophilic antioxidants might serve as indicators of lipoprotein oxidn. in the arterial wall and as markers of the development of atherosclerosis. However, it is unknown whether the measurement of plasma antioxidants is able to reflect atherogenesis or its risk. To assess whether the levels of lipophilic antioxidants in human plasma can discriminate between subjects with and without atherosclerosis, the authors measured the lipophilic antioxidants .alpha.-tocopherol, .**gamma.-tocopherol**, .alpha.-carotene, .beta.-carotene and ubiquinol-10 in plasma of 34 patients with coronary **heart disease** (CHD) and in 40 control subjects. The authors found that .alpha.-carotene and .**gamma.-tocopherol** were significantly lower in plasma of CHD patients compared to controls. This decrease was significantly independent of whether the antioxidants were expressed as its abs. amts. in plasma (for .alpha.-carotene, and for .**gamma.-tocopherol**) or normalized to plasma lipids (for both). In contrast, .beta.-carotene was only lower in plasma of CHD patients in comparison to controls, when normalized to the lipids. Independent contributions of different parameters to the variation in these plasma antioxidants were estd. using multiple regression approach. The anal. showed that both the decrease in .alpha.-carotene and the decrease in .**gamma.-tocopherol** were significantly assocd. only with the presence of CHD, while the decrease in .beta.-carotene was significantly related to the presence of hyperlipidemia. In striking contrast, no decrease in plasma .alpha.-tocopherol and ubiquinol-10 was detected in the patient group independently of how these antioxidants were expressed. These data suggest that plasma levels of .alpha.-carotene and .**gamma.-tocopherol** may represent markers of atherosclerosis in humans. Measuring these antioxidants may be of clin. importance as a practical approach to assess atherogenesis and/or its risk.

IT **7616-22-0**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(lipophilic antioxidants in blood plasma as markers of atherosclerosis in humans)

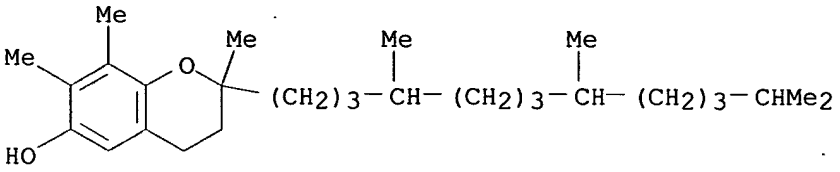
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 14-5 (Mammalian Pathological Biochemistry)
IT 7488-99-5, .alpha.-Carotene (natural) **7616-22-0**
RL: BOC (Biological occurrence); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(lipophilic antioxidants in blood plasma as markers of atherosclerosis in humans)

RE.CNT 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L46 ANSWER 16 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1999:60676 HCAPLUS
DN 130:235752
TI Consumption of vitamin E in coronary circulation in patients with variant angina
AU Miwa, Kunihisa; Igawa, Akihiko; Nakagawa, Keiko; Hirai, Tadakazu; Inoue, Hiroshi
CS Department of Internal Medicine, Toyama Medical and Pharmaceutical University, Toyama, 930-0194, Japan
SO Cardiovasc. Res. (1999), 41(1), 291-298
CODEN: CVREAU; ISSN: 0008-6363
PB Elsevier Science B.V.
DT Journal
LA English
AB The blood plasma status of vitamin E may be linked to the coronary **artery spasm**. This study was designed to det. whether vitamin E is actually consumed in the coronary circulation in patients with active variant (Prinzmetal) angina pectoris having repetitive spasm-induced transient myocardial ischemia and reperfusion. Blood samples were obtained simultaneously from the aortic root, coronary sinus, and right atrium in 12 patients with variant angina due to spasm of the left coronary artery, 9 patients with stable effort angina, and 9 control subjects. Plasma vitamin E (.alpha.- and .**gamma**.-**tocopherol**) concns. were detd. by HPLC and plasma lipid peroxides were measured as thiobarbituric acid-reactive substances (TBARS). At baseline, both plasma .alpha.- and .**gamma**.-**tocopherol** levels were lower in the coronary sinus (5.50.+-.0.50 and 0.55.+-.0.07 mg/L) than in the aortic root (6.63.+-.0.57 and 0.63.+-.0.08 mg/L) and also in the right atrium (6.44.+-.0.61 and 0.63.+-.0.09 mg/L) in the variant angina group. The TBARS levels were higher in the coronary sinus than in the aortic root in this group. The TBARS levels were not different between the samples from the coronary sinus and the aortic root or the right atrium in controls and in the stable effort angina group. The coronary sinus-aortic difference in plasma vitamin E levels in the variant angina group was not altered after left coronary **artery spasm** induced by intracoronary injection of acetylcholine. The plasma vitamin E levels in the aortic root, coronary sinus, and right atrium all remained unchanged in the stable effort angina group after the pacing-induced angina and in controls after intracoronary administration of acetylcholine. Thus, transcardiac decrease in blood plasma vitamin E concns. concomitant with lipid peroxide formation was demonstrated in patients with active variant angina, suggesting actual consumption of this major endogenous antioxidant. Oxidative stress and vitamin E exhaustion may be involved in the pathogenesis of coronary **artery spasm**.
IT **7616-22-0, .gamma. Tocopherol**
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(vitamin E uptake in heart coronary circulation in patients with variant angina)
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 14-5 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 18
IT 59-02-9, .alpha. Tocopherol 1406-18-4, Vitamin e **7616-22-0, .gamma. Tocopherol**
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(vitamin E uptake in heart coronary circulation in patients with variant angina)
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

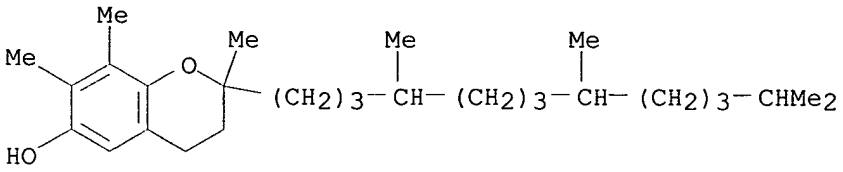
L46 ANSWER 17 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1998:635074 HCAPLUS
DN 129:341933
TI Antioxidant content in low density lipoprotein and lipoprotein oxidation in vivo and in vitro
AU Tertov, Vladimir V.; Sobenin, Igor A.; Kaplun, Victor V.; Orekhov, Alexander N.
CS Institute of Experimental Cardiology, Cardiology Research Center, Moscow, 121552, Russia
SO Free Radical Res. (1998), 29(2), 165-173
CODEN: FRARER; ISSN: 1071-5762
PB Harwood Academic Publishers
DT Journal
LA English
AB Human blood contains naturally occurring multiple-modified low d. lipoprotein (nomLDL) capable of inducing the accumulation of cholesteryl esters in the cells of human aortic intima. NomLDL is desialylated particles of small size with an increased electroneg. charge which can be sepd. from native low d. lipoprotein (LDL) by lectin chromatog. The purpose of this study was to det. the content of antioxidants in native and nomLDL obtained from healthy subjects and from patients with coronary **heart disease** as well as to elucidate a possible relationship between the level of antioxidants and the degree of in vivo and in vitro LDL oxidizability. The apoB-bound cholesterol level in native and nomLDL of healthy subjects was 0.25 .+-. 0.08 and 0.28 .+-. 0.05 mol/mol apoB, resp. The level of apoB-bound cholesterol in native LDL of coronary atherosclerosis patients showed no significant difference from that in healthy subjects' native lipoprotein. At the same time, the level of apoB-bound cholesterol in patients' nomLDL was 7-fold higher than in native LDL. The av. duration of the lag phase of native LDL oxidn. did not show a significant difference between the lipoprotein of healthy subjects and coronary atherosclerosis patients. The lag phase of nomLDL obtained from healthy subjects and patients was significantly shorter (3- and 6-fold, resp.) than for their native LDL. The latter finding points to their increased susceptibility to in vitro oxidn. Oxidizability of total LDL prepns. correlated pos. with their nomLDL content. The content of all the antioxidants studied (coenzyme-Q10, .alpha.- and .**gamma**.-**tocopherols**, .beta.-carotene and lycopene) in nomLDL was 1.5- to 2-fold lower than in native LDL. The level of apoB-bound cholesterol in nomLDL, correlated pos. with the ubiquinone-10 content and showed neg. correlation with ubiquinol-10 and .beta.-carotene levels. On the other

hand, the content of apoB-bound cholesterol in native LDL correlated pos. with the ubiquinol-10 level. Susceptibility of nomLDL to in vitro oxidn. exhibited neg. correlation with .alpha.-tocopherol and .beta.-carotene levels and a pos. correlation with the ubiquinone-10 content. On the contrary, oxidizability of native LDL correlated pos. with the ubiquinone-10 level. Conclusions: (a) elevated apoB-bound cholesterol level in nomLDL of coronary atherosclerosis patients indicates that peroxidn. of lipids occurs in vivo; (b) in vivo lipoperoxidn. in nomLDL is corroborated by increased proportion of oxidized form of coenzyme-Q10; (c) content of lipid-sol. antioxidants in nomLDL is lower than in native lipoprotein; (d) nomLDL has a higher susceptibility to in vitro oxidn. than native LDL; (e) it is necessary to use isolated subfractions of native LDL and nomLDL, but not total lipoprotein prepns., to study the mechanisms of lipid peroxidn.

IT **7616-22-0, .gamma.-Tocopherol**
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(antioxidant content in low d. lipoprotein and lipoprotein oxidn. in vivo and in vitro)

RN 7616-22-0 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 13-2 (Mammalian Biochemistry)
Section cross-reference(s): 14

IT 57-88-5, Cholesterol, biological studies 59-02-9, .alpha.-Tocopherol 303-98-0, Coenzyme-Q10 502-65-8, Lycopene 606-06-4, Ubiquinone-10 7235-40-7, .beta.-Carotene **7616-22-0, .gamma.-Tocopherol**
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(antioxidant content in low d. lipoprotein and lipoprotein oxidn. in vivo and in vitro)

L46 ANSWER 18 OF 32 HCAPLUS COPYRIGHT 2002 ACS

AN 1998:592780 HCAPLUS

DN 129:301980

TI Mechanisms of phytochemical inhibition of carcinogenesis: elucidating the role of **.gamma.-tocopherol** in nutrition

AU Burnett, T. S.; Tanaka, Y.; Harwood, P. J.; Cooney, R. V.

CS University of Hawaii Cancer Research Center, Honolulu, HI, 96813, USA

SO ACS Symp. Ser. (1998), 701(Functional Foods for Disease Prevention I: Fruits, Vegetables, and Teas), 45-58
CODEN: ACSMC8; ISSN: 0097-6156

PB American Chemical Society

DT Journal; General Review

LA English

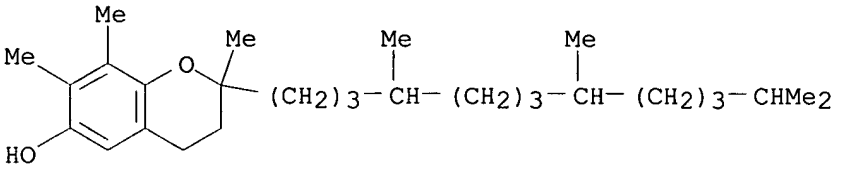
AB A review with 41 refs. Epidemiol. studies have consistently demonstrated a protective effects of fruit and vegetable consumption against the development of many forms of cancer and **heart disease**.

Identifying the responsible chem. constituents and their mechanism(s) of action are crit. Recent studies have found that blood serum .**gamma.-tocopherol** levels are inversely related to risk for cardiovascular disease and some cancers, despite the fact that sources of .**gamma.-tocopherol** are limited to dietary oils of plant origin. Tocopherols are often highly concd. in the germ plasm of seeds and esp. high concns. of .**gamma.-tocopherol** are in the germ of peanuts. Whereas .alpha.-tocopherol is the most biopotent of the vitamin E analogs, .**gamma.-tocopherol** is more effective in preventing neoplastic transformation and cellular damage induced by cytokines. The mechanism of .**gamma.-tocopherol** action in preventing cellular damage may be unique relative to other phytochems. and potentially related to specific chem. and biol. properties of .**gamma.-tocopherol**, including the ability to chem. reduce NO2 to NO, enhance cellular NO synthesis, alter the kinetics of cell growth, enhance cell satn. d., and reduce DNA strand breaks in C3H 10T1/2 murine fibroblasts.

IT **7616-22-0, .gamma. Tocopherol**
RL: BAC (Biological activity or effector, except adverse); FFD (Food or feed use); BIOL (Biological study); USES (Uses)
(dietary .**gamma.-tocopherol** mechanism of inhibition of carcinogenesis and cardiovascular disease)

RN 7616-22-0 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 18-0 (Animal Nutrition)
Section cross-reference(s): 14

ST review nutrition **gamma tocopherol** cancer prevention

IT Cardiovascular diseases
Nutrition (animal)
Tumors (animal)
(dietary .**gamma.-tocopherol** mechanism of inhibition of carcinogenesis and cardiovascular disease)

IT **7616-22-0, .gamma. Tocopherol**
RL: BAC (Biological activity or effector, except adverse); FFD (Food or feed use); BIOL (Biological study); USES (Uses)
(dietary .**gamma.-tocopherol** mechanism of inhibition of carcinogenesis and cardiovascular disease)

L46 ANSWER 19 OF 32 HCAPLUS COPYRIGHT 2002 ACS

AN 1998:169475 HCAPLUS

DN 128:248580

TI Association of NO synthase inhibitors with trappers of reactive oxygen species

IN Chabrier De Lassauniere, Pierre-Etienne; Bigg, Denis

PA Societe De Conseils De Recherches Et D'applications Scientifiques (S.C.R.A.S, Fr.; Chabrier De Lassauniere, Pierre-Etienne; Bigg, Denis

SO PCT Int. Appl., 22 pp.
CODEN: PIXXD2

DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9809653	A1	19980312	WO 1997-FR1567	19970905
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	RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	FR 2753098	A1	19980313	FR 1996-10875	19960906
	FR 2753098	B1	19981127		
	AU 9742111	A1	19980326	AU 1997-42111	19970905
	AU 734296	B2	20010607		
	EP 939654	A1	19990908	EP 1997-940183	19970905
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2000517336	T2	20001226	JP 1998-512314	19970905
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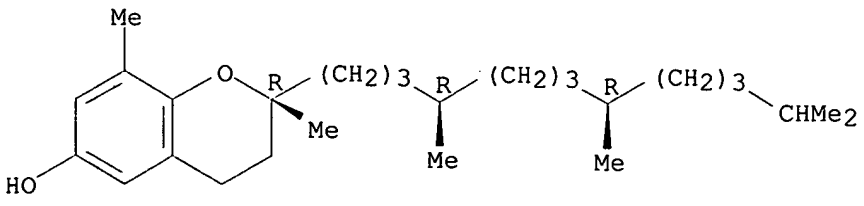
	NO 9901100	A	19990505	NO 1999-1100	19990305
PRAI	FR 1996-10875	A	19960906		
	WO 1997-FR1567	W	19970905		

AB The invention concerns a pharmaceutical compn. contg., as active principle, at least one NO synthase-inhibiting substance and at least one reactive oxygen-trapping substance, optionally with a pharmaceutically acceptable support. The invention also concerns a product contg. at least one NO synthase-inhibiting substance and at least one reactive oxygen-trapping substance as combined product of these active principles in sep. form.

IT **119-13-1, .delta.-Tocopherol 148-03-8, .beta.-Tocopherol 7616-22-0, .gamma.-Tocopherol**
RL: BAC (Biological activity or effector, except adverse); PEP (Physical, engineering or chemical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(assocn. of NO synthase inhibitors with trappers of reactive oxygen species)

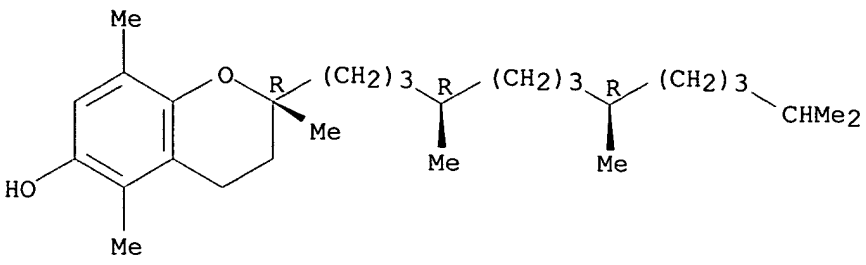
RN 119-13-1 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,8-dimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

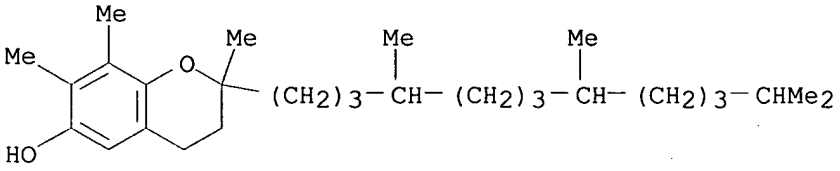


RN 148-03-8 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,5,8-trimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)-rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



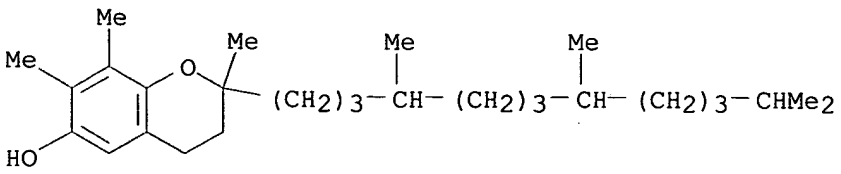
IC ICM A61K045-06
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1
IT AIDS (disease)
Airway inflammation
Alcoholism
Amyloidosis
Anti-inflammatory drugs
Anti-ischemic agents
Antiarthritics
Antiatherosclerotics
Anticonvulsants
Antidepressants
Antidiabetic agents
Antidiarrheals
Antiemetics
Antihypertensives
Antimigraine drugs
Antioxidants (pharmaceutical)
Antiproliferative agents
Antipsychotics
Antithrombotics
Antitumor agents
Antiviral agents
Anxiety
Anxiolytics
Atherosclerosis
Autoimmune diseases
Cardiovascular agents
Cardiovascular diseases
Cerebral hemorrhage
Cerebral ischemia

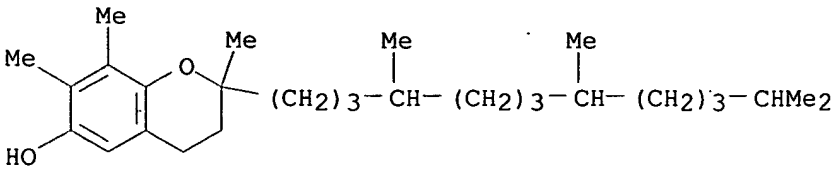
Cerebrovascular diseases
Cognitive disorders
Depression (mental)
Diabetes mellitus
Diarrhea
Drug delivery systems
Dyspepsia
Epilepsy
Fibrosis
Glomerulonephritis
Hypertension
Immunomodulators
Inflammation
Lupus erythematosus
Migraine
Myocardial infarction
Nervous system agents
Nervous system diseases
Parasiticides
Portal hypertension
Psoriasis
Pulmonary hypertension
Pulmonary inflammation
Radioprotectants
Reperfusion
Reproductive disorders
Rheumatoid arthritis
Schizophrenia
Septic shock
Sleep disorders
Solar radiation
Thrombosis
Transplant (organ)
Tumors (animal)
Viral infection
Vomiting
(assocn. of NO synthase inhibitors with trappers of reactive oxygen species)

IT 50-81-7, Ascorbic acid, biological studies 56-87-1D, Lysine, derivs. 59-02-9, .alpha.-Tocopherol 70-26-8D, Ornithine, derivs. 73-31-4, Melatonin 74-79-3D, L-Arginine, derivs. 79-17-4, Aminoguanidine 97-53-0, Eugenol **119-13-1**, **.delta.-Tocopherol** 121-79-9, n-Propyl gallate **148-03-8**, **.beta.-Tocopherol** 149-91-7, Gallic acid, biological studies 288-32-4, Imidazole, biological studies 303-98-0, Coenzyme q10 306-60-5, Agmatine 331-39-5, Caffeic acid 489-01-0, 2,6-Di-tert-butyl-4-methoxyphenol 490-23-3, .epsilon.-Tocopherol 530-59-6, Sinapinic acid 616-91-1, N-Acetyl cysteine 1421-49-4, 3,5-Di-tert-butyl-4-hydroxybenzoic acid 1848-68-6 2149-70-4, Nitroarginine 2154-67-8 2214-67-7 2226-96-2, Tempol 2942-42-9, 7-Nitroindazole 2986-20-1, S-Ethylisothiourea 3737-39-1 5401-94-5, 5-Nitroindazole 7235-40-7, .beta.-Carotene 7597-18-4, 6-Nitroindazole **7616-22-0**, **.gamma.-Tocopherol** 17035-90-4 21598-06-1, 5-Hydroxyindole-2-carboxylic acid 22780-54-7, 2-Iminopiperidine 23288-49-5, Probucol 25371-96-4, 1,2-(Trifluoromethylphenyl)imidazole 30480-64-9 41078-65-3 50903-99-6, L-Ornithine, N5-[imino(nitroamino)methyl]-, methyl ester 51481-61-9, Cimetidine 52602-39-8, 4-Hydroxycarbazole 53188-07-1, Trolox 72956-09-3, Carvedilol 156719-41-4, S-Methyl-L-thiocitrulline 158875-72-0,

S-Ethyl-L-thiocitrulline 171082-82-9 179555-23-8 204771-24-4
204866-75-1, .tau.-Tocopherol
RL: BAC (Biological activity or effector, except adverse); PEP (Physical, engineering or chemical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(assocn. of NO synthase inhibitors with trappers of reactive oxygen species)

L46 ANSWER 20 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1998:41352 HCAPLUS
DN 128:166737
TI Enhanced plasma level of lipid peroxidation in Iranians could be improved by antioxidants supplementation
AU Meraji, S.; Ziouzenkova, O.; Resch, U.; Khoschsorur, A.; Tatzber, F.; Esterbauer, H.
CS Cardiovascular Research Institute, Tehran, Iran
SO Eur. J. Clin. Nutr. (1997), 51(5), 318-325
CODEN: EJCNEQ; ISSN: 0954-3007
PB Stockton Press
DT Journal
LA English
AB The effects of dietary supplementation with antioxidants on factors that may increase the risk of coronary **heart disease** (CHD) were studied in 21 Iranian men. One group received 30 mg .beta.-carotene/d plus placebo for .alpha.-tocopherol; the other group received .beta.-carotene plus 400 IU .alpha.-tocopherol for 10 wk. The concns. of antioxidants in blood plasma and low-d. lipoproteins (LDL), plasma lipid profile, autoantibody against oxidized LDL (OLAb) and malondialdehyde (MDA) concns. in plasma were measured. Oxidative resistance of LDL was estd. using conjugated diene assay. The Iranians had lower plasma levels of total cholesterol, LDL-cholesterol, and HDL-cholesterol compared to healthy Austrian controls. Although the baseline concns. of .alpha.-tocopherol and .beta.-carotene were comparable with Austrians, lycopene, canthaxanthin and lutein levels were higher in Iranians. In vitro oxidative resistance of LDL, measured as lag-time, was slightly higher in Iranians compared to Austrians. Plasma MDA and OLAb concns. were higher in Iranians. Both dietary supplementations reduced the plasma MDA concns. The combined supplement increased OLAb concns. as well as the oxidn. lag-time. Thus, high plasma MDA levels of Iranians can be decreased by dietary .beta.-carotene supplementation with or without .alpha.-tocopherol. However, .alpha.-tocopherol is a more powerful antioxidant which can increase the resistance of LDL to oxidn., reduce the MDA concns. in blood plasma and increase the levels of autoantibodies to oxidized LDL.
IT **7616-22-0, .gamma.-Tocopherol**
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(blood plasma lipid peroxidn. in Iranian men given antioxidant supplements of .beta.-carotene and .alpha.-tocopherol)
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)





CC 18-2 (Animal Nutrition)
IT 57-88-5, Cholesterol, biological studies 68-26-8, Retinol 127-40-2, Lutein 144-68-3, Zeaxanthin 432-70-2, .alpha. Carotene 472-70-8, .beta. Cryptoxanthin 502-65-8, Lycopene 514-78-3, Canthaxanthin **7616-22-0, .gamma.-Tocopherol**
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(blood plasma lipid peroxidn. in Iranian men given antioxidant supplements of .beta.-carotene and .alpha.-tocopherol)

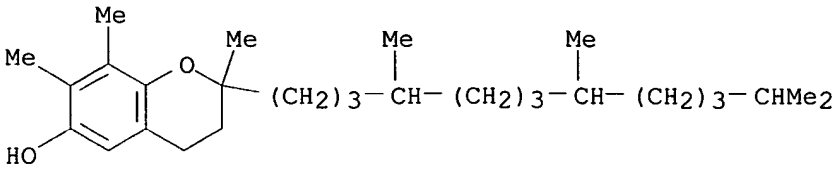
L46 ANSWER 21 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1997:778055 HCAPLUS
DN 128:46708
TI Antioxidants in adipose tissue and myocardial infarction in a Mediterranean area. The EURAMIC study in Malaga
AU Gomez-Aracena, J.; Sloots, L.; Garcia-Rodriguez, A.; Van't Veer, P.; Gomez-Gracia, E.; Garcia-Alcantara, A.; Martin-Moreno, J. M.; Kok, F. J.; Navajas, J. Fernandez-Crehuet
CS Department of Preventive Medicine and Public Health, University of Malaga and Hospital Clinico Universitario, Malaga, 29071, Spain
SO Nutr., Metab. Cardiovasc. Dis. (1997), 7(5), 376-382
CODEN: NMCDEE; ISSN: 0939-4753
PB Medikal Press
DT Journal
LA English
AB Many studies have suggested that the Mediterranean diet has a protective effect against coronary **heart disease**. One of the explanations is the high content of antioxidants which might protect LDL particles from oxidn. and development of atherosclerosis. As part of the EURAMIC Study, a multicenter case-control study, the relationship between antioxidants in adipose tissue and first acute myocardial infarction was investigated in 100 cases and 102 controls living in Malaga, Spain. Tocopherol, .beta.-carotene, **.gamma.-tocopherol**, .alpha.-carotene, lycopene and retinol were measured and expressed in .mu.g/g fatty acids. Mean levels of antioxidants for cases vs. controls, were as follows: .alpha.-tocopherol, 234.7 in cases and 196.6 in controls; **.gamma.-tocopherol** concn., 17.8 in cases and 17.1 in controls; .beta.-carotene, 0.21 and 0.26 in controls; .alpha.-carotene, 0.04 in cases and 0.05 in controls; retinol, 1.07 in cases and 1.28 in controls; and lycopene, 0.25 in both cases and controls. Neg. correlations were found between alc. intake and .alpha.-tocopherol, **.gamma.-tocopherol** and .beta.-carotene in controls, whereas no such correlation was found in cases. Body mass index was also inversely correlated with .beta.-carotene, .alpha.-carotene, and lycopene in controls; in cases this factor was likewise found inversely correlated with .beta.-carotene, .alpha.-carotene and lycopene. The OR for risk of myocardial infarction in the lowest vs. the highest quintile of lycopene concn., adjusted for age, family history of coronary **heart disease** and cigarette smoking, was 2.55. For retinol this OR was 2.97. No assocns. between .alpha.-tocopherol, **.gamma.-tocopherol**, .beta.-carotene or .alpha.-carotene and MI were obsd. This study provides evidence that retinol and lycopene may play a

protective role in MI but the possibility that these nutrients might only be just dietary markers should not be excluded. Furthermore the results do not suggest that other antioxidants are assocd. with myocardial infarction in our area.

IT **7616-22-0, .gamma.-Tocopherol**
RL: BOC (Biological occurrence); FFD (Food or feed use); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(antioxidants in adipose tissue and myocardial infarction in Mediterranean area)

RN 7616-22-0 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 14-5 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 4, 18

IT 59-02-9, .alpha.-Tocopherol 68-26-8, Retinol 432-70-2, .alpha.-Carotene 502-65-8, Lycopene 7235-40-7, .beta.-Carotene **7616-22-0, .gamma.-Tocopherol**
RL: BOC (Biological occurrence); FFD (Food or feed use); BIOL (Biological study); OCCU (Occurrence); USES (Uses)
(antioxidants in adipose tissue and myocardial infarction in Mediterranean area)

L46 ANSWER 22 OF 32 HCAPLUS COPYRIGHT 2002 ACS

AN 1997:684290 HCAPLUS

DN 127:336651

TI Unit dosage forms containing magnesium, vitamin C, vitamin E, folate and selenium for treatment of vasoconstriction and related conditions

IN Richardson, Kenneth T.; Pearson, Don C.

PA Richell Laboratories L.L.C., USA; Richardson, Kenneth T.; Pearson, Don C.

SO PCT Int. Appl., 23 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9737670	A1	19971016	WO 1997-US4286	19970318
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9725833	A1	19971029	AU 1997-25833	19970318
	US 5849338	A	19981215	US 1997-849068	19970826
	US 6042849	A	20000328	US 1998-111055	19980707
PRAI	US 1996-15115P	P	19960410		

US 1996-753967 A2 19961204
WO 1997-US4286 W 19970318

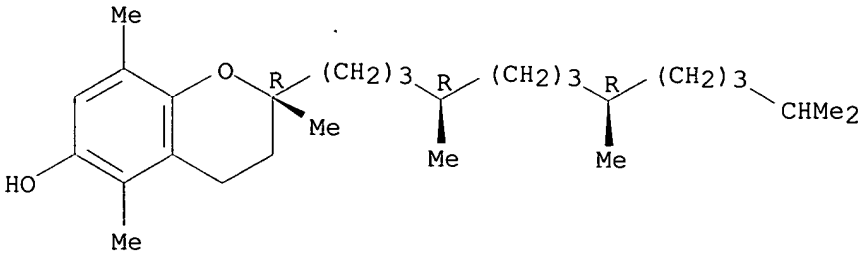
AB Magnesium is formulated in combination with vitamin E, vitamin C, folate, selenium, and optionally melatonin in a unit dosage form for oral administration, for the treatment of vasoconstriction and the physiol. and pathol. conditions giving rise to vasoconstriction. These active agents complement each other in suppressing these conditions, using a variety of mechanisms operating in conjunction with one another. The inclusion of magnesium in a plurality of forms provides addnl. advantages in terms of controlling and sustaining the release of magnesium in locations along the digestive tract where the magnesium has its greatest effectiveness as a therapeutic agent, thus improving control over the clin. bioavailability of magnesium and in improving the selection of appropriate therapeutic ranges. A tablet was formulated contg. Mg acetate tetrahydrate 67.67, Mg ascorbate 64.17, Mg citrate pentahydrate 54.36, MgO 118.54, Mg stearate 3.55, selenophenol or selenomethanol 0.1, melatonin 0.1-40, folic acid 0.2, starch 120 mg, and tocopherol succinate 60 IU. The tablet was further coated with Opadry.

IT **148-03-8, .beta.-Tocopherol**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oral dosage forms contg. minerals and vitamins for treatment of vasoconstriction and related conditions)

RN 148-03-8 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,5,8-trimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)-rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



IC ICM A61K033-06
ICS A61K033-06; A61K033-04; A61K031-505; A61K031-375; A61K031-355

CC 63-6 (Pharmaceuticals)

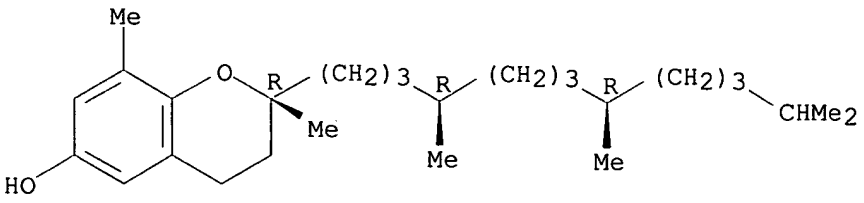
IT Capsules (drug delivery systems)
Tablets (drug delivery systems)
Vasoconstriction
Vasospasm
(oral dosage forms contg. minerals and vitamins for treatment of vasoconstriction and related conditions)

IT 50-81-7, Ascorbic acid, biological studies 58-95-7, .alpha.-Tocopherol acetate 59-02-9, .alpha.-Tocopherol 59-30-3, Folic acid, biological studies 73-31-4, Melatonin 142-72-3, Magnesium acetate **148-03-8, .beta.-Tocopherol** 557-04-0, Magnesium stearate 645-96-5, Selenophenol 869-06-7, Magnesium malate 1309-48-4, Magnesium oxide, biological studies 4345-03-3, .alpha.-Tocopherol succinate 6486-05-1, Selenomethanol 7779-25-1, Magnesium citrate 7782-49-2, Selenium, biological studies 14783-68-7 15431-40-0, Magnesium ascorbate 18917-93-6, Magnesium lactate 34717-03-8, Magnesium orotate
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(oral dosage forms contg. minerals and vitamins for treatment of vasoconstriction and related conditions)

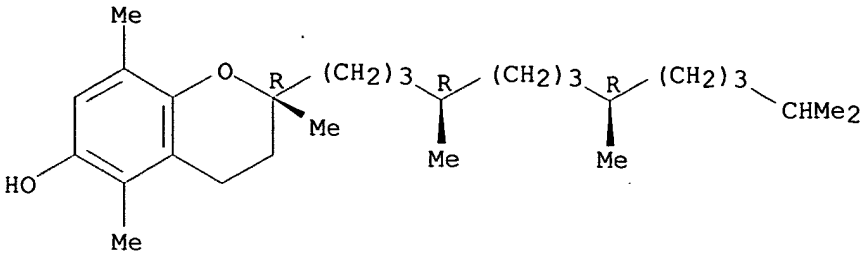
L46 ANSWER 23 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1997:458709 HCAPLUS
DN 127:80509
TI Investigation on antioxidant and antiatherosclerosis components in plants. I. The plum of Briancon
AU Rousseau, Laurence; Villet, Annick; Ravel, Anne; Alary, Josette
CS Lab. Chimie Analytique, GREPO, UFR Pharmacie Grenoble, La Tronche, 38700, Fr.
SO Ann. Falsif. Expert. Chim. Toxicol. (1996), 89(937), 235-245
CODEN: AFETDF; ISSN: 0242-6110
PB Societe des Experts-Chimistes de France
DT Journal
LA French
AB The fruit of the plum tree of Briancon (also known as Afatoulier) was studied. This study was mainly concerned with the anal. of the pulp and the oil extd. from the kernel. The pulp was characterized by a low concn. of sugars, acidity, and presence of polyphenols and fiber (of which one part was in the form of pectins). The plum of Briancon can therefore be used for the manuf. of health foods (most probably jellies). Pectins would facilitate the prepn. of jams and polyphenols and fiber would be beneficial for the health. The consumption of the raw fruit is not envisaged due to its acidity. The oil had a compn. similar to that of olive oil and therefore could be used in food technol. for seasoning and frying. The oil was rich in **.gamma.-tocopherols** which ensure its antioxidant stability and offers a nutritional value. The pulp and oil of the plum of Briancon could help in the prevention of cancer and **heart diseases.**
IT **119-13-1, .delta.-Tocopherol 148-03-8, .beta.-Tocopherol 7616-22-0, .gamma.-Tocopherol**
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(antioxidant and antiatherosclerosis components in plum of Briancon)
RN 119-13-1 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,8-dimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

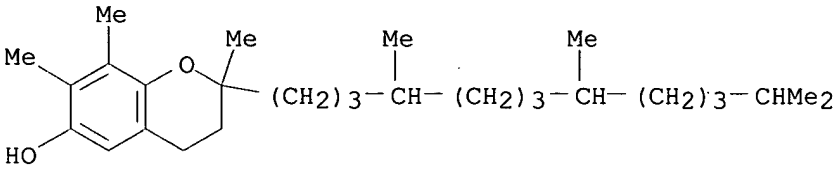


RN 148-03-8 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,5,8-trimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)-rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 17-10 (Food and Feed Chemistry)
IT 50-81-7, Vitamin C, biological studies 57-10-3, Hexadecanoic acid, biological studies 57-11-4, Octadecanoic acid, biological studies 57-88-5, Cholesterol, biological studies 59-02-9, .alpha.-Tocopherol 60-33-3, 9,12-Octadecadienoic acid (Z,Z)-, biological studies 68-26-8, Retinol 83-46-5, .beta.-Sitosterol 83-48-7, Stigmasterol 112-80-1, Oleic acid, biological studies 112-85-6, Docosanoic acid **119-13-1**, **.delta.-Tocopherol** **148-03-8**, **.beta.-Tocopherol** 373-49-9, Palmitoleic acid 463-40-1 474-62-4, Campesterol 506-12-7, Heptadecanoic acid 506-30-9, Arachidic acid 544-63-8, Tetradecanoic acid, biological studies 1981-50-6 5561-99-9, Gondoic acid 7440-09-7, Potassium, biological studies **7616-22-0**, **.gamma.-Tocopherol** 9000-69-5, Pectins 18472-36-1, .DELTA.5-Avenasterol
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(antioxidant and antiatherosclerosis components in plum of Briancon)

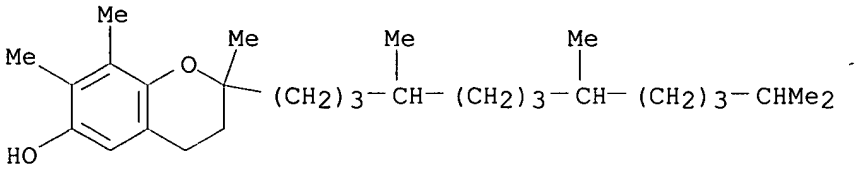
L46 ANSWER 24 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1996:685803 HCAPLUS
DN 126:7007
TI Do hydroxy-carotenoids prevent coronary **heart disease**? A comparison between Belfast and Toulouse
AU Howard, A. N.; Williams, N. R.; Palmer, C. R.; Cambou, J. P.; Evans, A. E.; Foote, J. W.; Marques-Vidal, P.; McCrum, E. E.; Ruidavets, J. B.; et al.
CS Dep. Pathol., Papworth Hosp. NHS Trust, Cambridge, CB3 8RE, UK
SO Int. J. Vitam. Nutr. Res. (1996), 66(2), 113-118
CODEN: IJVNAP; ISSN: 0300-9831
PB Hogrefe & Huber
DT Journal
LA English
AB High intakes of antioxidants in fruit, vegetables and wine are thought to protect against coronary **heart disease** (CHD). Because people in Toulouse have a much lower incidence of CHD compared with

Belfast, the plasma concns. of antioxidant vitamins and carotenoids in the two populations have been compared. The major difference was in some of the plasma carotenoids. Hydroxy-carotenoids were twice as high in Toulouse in both sexes, notably lutein which occurs principally in dark green vegetables and .beta.-cryptoxanthin which occurs chiefly in citrus fruits. In addn., .alpha.-carotene was 50% higher in Toulouse, .**gamma.-tocopherol** was 50% higher in Belfast. Other plasma vitamins and carotenoids were not significantly different. If antioxidants play a role in preventing CHD, then the hydroxy-carotenoids are major candidates for further investigation.

IT **7616-22-0, .gamma.-Tocopherol**
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(hydroxy-carotenoids in prevention of coronary **heart disease**)

RN 7616-22-0 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 18-2 (Animal Nutrition)
Section cross-reference(s): 13

ST carotenoid plasma **heart disease**

IT Vitamins
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(antioxidant; hydroxy-carotenoids in prevention of coronary **heart disease**)

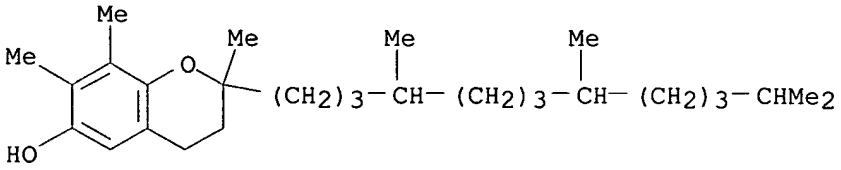
IT Coronary artery disease
Plasma (blood)
(hydroxy-carotenoids in prevention of coronary **heart disease**)

IT Apolipoprotein A-I
Apolipoprotein B
Carotenes, biological studies
Glycerides, biological studies
High-density lipoproteins
Lipoproteins
Low-density lipoproteins
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(hydroxy-carotenoids in prevention of coronary **heart disease**)

IT 57-88-5, Cholesterol, biological studies 59-02-9, .alpha.-Tocopherol 68-26-8, Retinol 127-40-2, Lutein 472-70-8, .beta.-Cryptoxanthin 502-65-8, Lycopene 7235-40-7, .beta.-Carotene 7488-99-5, .alpha.-Carotene **7616-22-0, .gamma.-Tocopherol** 24480-38-4, .alpha.-Cryptoxanthin
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(hydroxy-carotenoids in prevention of coronary **heart**

disease)

L46 ANSWER 25 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1996:150619 HCAPLUS
DN 124:259498
TI Fatty acid composition of cholesterol esters and serum tocopherols in Melanesians apparently free from cardiovascular disease: the Kitava study
AU Lindeberg, S.; Vessby, B.
CS Department of Community Health Sciences, Lund University, Swed.
SO Nutr., Metab. Cardiovasc. Dis. (1995), Volume Date 1995, 5(1), 45-53
CODEN: NMCDEE; ISSN: 0939-4753
DT Journal
LA English
AB Fatty acid (FA) compn. of cholesterol esters (CE) and serum tocopherols were measured in 168 subsistence horticulturalists of Kitava, Trobriand Islands, Papua New Guinea, whose diet consists of tubers, fruit, coconut, fish and vegetables with a negligible influence of western food. Stroke and ischemic **heart disease** (IHD) appear to be absent despite high smoking rates and intermediate serum lipoprotein levels. Comparisons were made with serum samples from healthy Swedish subjects, randomly selected from employees of a telephone company. A dietary survey was made in Kitava including diet history and weighing of constituents of ready-to-eat portions. The percentages of all CE-FAs except arachidonic and oleic acid differed markedly between the two populations. Kitavans had higher satd. FAs while polyunsatd. FAs (PUFAs) were lower. Lauric acid was only detectable in trace amts. despite a very high estd. intake in Kitava. In spite of a lower intake, palmitic acid was higher in Kitavans, possibly reflecting endogenous fat synthesis due to low total fat intake. Marine n-3 PUFAs were much higher while linoleic acid was much lower in Kitavans. Alpha tocopherol was slightly higher in Kitavan than in Swedish males, while it did not differ among females. **Gamma Tocopherol** was much lower in Kitavans. In conclusion, the high intake of marine n-3 PUFAs and the high n-3/n-6 ratio may partially explain the apparent absence of IHD in Kitava, while serum tocopherols in this study seem of little importance.
IT **7616-22-0, .gamma.-Tocopherol**
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(fatty acid compn. of cholesterol esters and serum tocopherols in New Guinea Melanesians apparently free from cardiovascular disease)
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



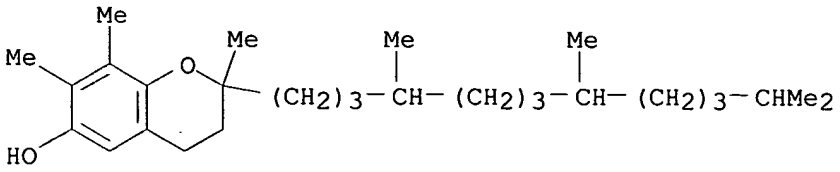
CC 18-5 (Animal Nutrition)
Section cross-reference(s): 13
IT 57-10-3, Palmitic acid, biological studies 57-88-5D, Cholesterol, esters 59-02-9, .alpha.-Tocopherol 60-33-3, Linoleic acid, biological studies 143-07-7, Lauric acid, biological studies 506-32-1, Arachidonic acid **7616-22-0, .gamma.-Tocopherol**

RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(fatty acid compn. of cholesterol esters and serum tocopherols in New Guinea Melanesians apparently free from cardiovascular disease)

L46 ANSWER 26 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1996:110052 HCAPLUS
DN 124:174392
TI Vitamin E level of plasma and erythrocyte membrane of the children from southern Keshan disease area
AU Liu, Zhongying; Li, Tiyuan; Liu, Jian; Jiang, Xiluo; An, Ruguo
CS Norman Bethune Univ. of Medical Science, Changchun, 130021, Peop. Rep. China
SO Yingyang Xuebao (1995), 17(4), 425-7
CODEN: YYHPA4; ISSN: 0512-7955
DT Journal
LA Chinese
AB Vitamin E, esp. .alpha.-tocopherol, level of plasma and erythrocyte membrane of the children from southern Keshan disease area is significantly lower than that of the control non-Keshan disease area. . **gamma.-Tocopherol** level in also lower in the children of the southern Keshan disease area.
CC 18-2 (Animal Nutrition)
IT **Heart, disease**
(Keshan, children of southern area of; vitamin E level of plasma and erythrocyte membrane of children from southern Keshan disease area)

L46 ANSWER 27 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1995:505376 HCAPLUS
DN 122:287931
TI Plasma fibrinogen, fibrinolysis and (Pro)vitamins; is there a connection?
AU Eliasson, M.; Asplund, K.; Evrin, P.-E.; Huhtasaari, F.; Johansson, I.
CS Dep. Med., Luleaa Hosp., Swed.
SO Fibrinolysis (1995), 9(2), 87-92
CODEN: FBRIE7; ISSN: 0268-9499
DT Journal
LA English
AB To investigate whether the relationship between anti-oxidant vitamins and cardiovascular disease can be mediated by influencing hemostasis or fibrinolysis. Cross-sectional population study. Population screening in the northern Sweden MONICA study. 102 Mean aged 40-49 yr, randomly selected. Univariate and multivariate relationships between on the one hand plasma fibrinogen, tPA activity and PAI-1 activity and on the other hand plasma levels of retinol, .beta.-carotene, vitamin C, .alpha.- and . **gamma.-tocopherol**. Plasma fibrinogen levels were inversely correlated to lipid-standardized retinol; a relationship that persisted after adjustment for possible confounders. TPA activity was directly related to .beta.-carotene and inversely to retinol (with or without lipid-standardization). In multiple regression anal., lipid-standardized retinol was still a significant predictor of tPA activity when possible confounders and PAI-1 activity were taken into consideration. PAI-1 activity correlated to retinol and inversely to .beta.-carotene but these (pro)vitamins were not significant predictors of PAI-1 activity when adjusted for confounders. High plasma retinol levels are assocd. with low plasma fibrinogen and impaired fibrinolytic activity. Other anti-oxidant (pro)vitamins seem not to act by influencing hemostasis for fibrinolysis.
IT **7616-22-0, .gamma.-Tocopherol**
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU

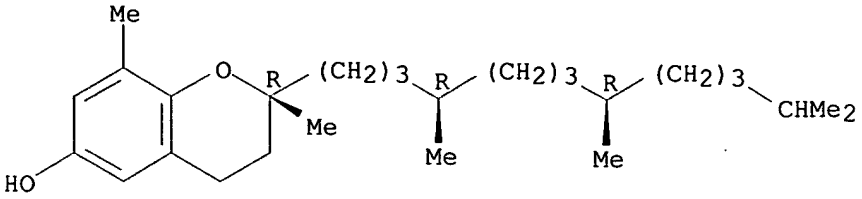
(Occurrence)
(fibrinogen, provitamins, plasminogen activator and inhibitor in human plasma in relation to myocardial infarction risk fibrinogen, provitamins, plasminogen activator and inhibitor in human plasma in relation to myocardial infarction risk)
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 14-5 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 8
IT **Heart, disease**
(infarction, fibrinogen, provitamins, plasminogen activator and inhibitor in human plasma in relation to myocardial infarction risk)
IT **7616-22-0, .gamma.-Tocopherol**
RL: BOC (Biological occurrence); BIOL (Biological study); OCCU (Occurrence)
(fibrinogen, provitamins, plasminogen activator and inhibitor in human plasma in relation to myocardial infarction risk fibrinogen, provitamins, plasminogen activator and inhibitor in human plasma in relation to myocardial infarction risk)

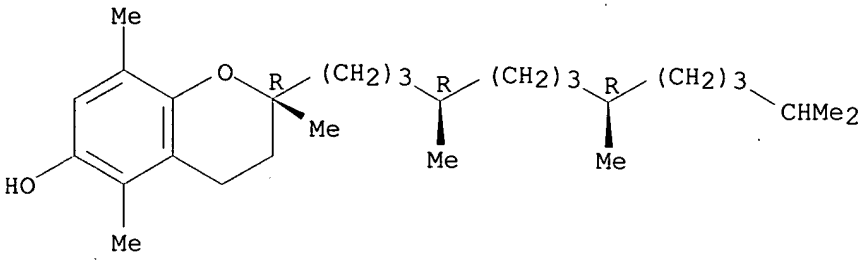
L46 ANSWER 28 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1992:19114 HCAPLUS
DN 116:19114
TI Age-related tocopherol content of normal and ischemic heart and liver of rats
AU Paranich, A. V.; Chaikina, L. A.
CS Kharkov State Univ., Kharkov, USSR
SO Fiziol. Zh. (Kiev) (1991), 37(5), 16-19
CODEN: FIZHDO; ISSN: 0201-8489
DT Journal
LA Russian
AB Expts. were conducted on the heart and liver of 1-, 3-, 12- and 24-mo-old rats in order to study the effect of ischemia of 1 h duration on the tocopherol content in these tissues. The parameters under study were shown to reliably decrease in all the cases. In the liver the content of all tocopherols decreased most rapidly in 1-mo-old rats. In the heart the most rapid decrease in the content of tocopherols was obsd. in 3-mo-old rats. The results obsd. permit estn. of the contribution of certain vitamins to repair of injuries caused by ischemia.
IT **119-13-1 148-03-8 7616-22-0**
RL: BIOL (Biological study)
(of heart and liver, ischemia and senescence effects on)
RN 119-13-1 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,8-dimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

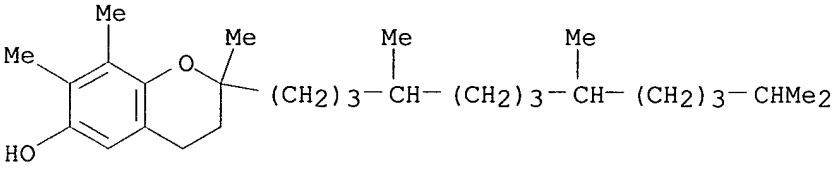


RN 148-03-8 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,5,8-trimethyl-2-[(4R,8R)-4,8,12-trimethyltridecyl]-, (2R)-rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 14-5 (Mammalian Pathological Biochemistry)
IT **Heart, disease**
(ischemia, tocopherol of heart response to, senescence effect on)
IT 59-02-9 **119-13-1 148-03-8 7616-22-0**
RL: BIOL (Biological study)
(of heart and liver, ischemia and senescence effects on)

L46 ANSWER 29 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1991:598523 HCAPLUS
DN 115:198523
TI Preparation of tocotrienols for the treatmentt of hypercholesterolemia, hyperlipidemia and **thromboembolic** disorders
IN Wright, John J.; Pearce, Bradley C.; Parker, Rex; Quereshi, Asaf A.
PA Bristol-Myers Co., USA
SO Eur. Pat. Appl., 30 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 2
PATENT NO. KIND DATE APPLICATION NO. DATE

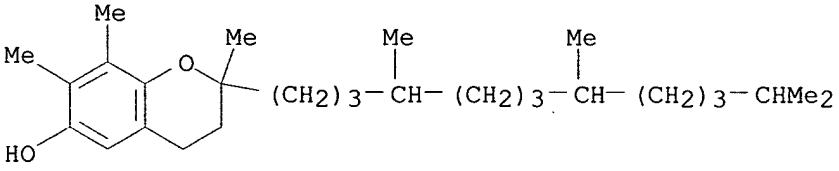
PI	EP 421419	A2	19910410	EP 1990-119040	19901004
	EP 421419	A3	19920401		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE				
	HU 54887	A2	19910429	HU 1990-6297	19901002
	HU 207442	B	19930428		
	ZA 9007906	A	19910626	ZA 1990-7906	19901003
PRAI	US 1989-416910		19891004		

AB Tocotrienol, .gamma.-tocotrienol and .delta.-tocotrienol are prepd. from natural sources and chem. synthesis to lower serum cholesterol, LDL-cholesterol, thromboxane A2, platelet factor 4, and platelet aggregation to ADP, epinephrine and collagen for treating hypercholesterolemia, hyperlipidemia, and **thromboembolic** disorders in birds and mammals. Prepn. of e.g. d,l-.gamma.-tocotrienol is described. Effectiveness of .gamma.-tocotrienol in lowering serum cholesterol level, etc. in humans, swine, and chickens was examd.

IT **7616-22-0, .gamma.-Tocopherol**
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(cholesterol synthesis inhibitory activity of)

RN 7616-22-0 HCAPLUS

CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



IC ICM A61K031-355

CC 1-8 (Pharmacology)
Section cross-reference(s): 26, 27

ST tocotrienol cholesterol lowering; hypercholesterolemia hyperlipidemia **thromboembolic** disorder tocotrienol

IT **Embolism**
(**thrombo-**, treatment of, tocotrienols for)

IT **7616-22-0, .gamma.-Tocopherol**
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(cholesterol synthesis inhibitory activity of)

IT 135897-88-0P 135897-90-4P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction of, in prepn. of tocotrienol for teating hypercholesterolemia, hypolipidemia and **thromboembolic** disorders)

IT 608-43-5P 135897-84-6P 135897-86-8P 135897-87-9P 135897-89-1P
135897-91-5P 135897-92-6P 135897-93-7P 135897-95-9P 135897-96-0P
135897-97-1P 135897-98-2P 135897-99-3P 135898-01-0P 135898-02-1P
135898-03-2P 135898-04-3P 135898-05-4P 135898-06-5P 136774-63-5P
136774-64-6P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation)
(prepn. and reaction of, in prepn. of tocotrienol for treating hypercholesterolemia, hyperlipidemia and **thromboembolic** disorders)

IT 47686-42-0P

RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of, in prepn. of tocotrienol for teating hypercholesterolemia, hypolipidemia and **thromboembolic** disorders)

IT 135970-14-8P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of, in prepn. of tocotrienol for treating hypercholesterolemia, hyperlipidiemia and **thromboembolic** disorders)

IT 20260-53-1, Nicotinyl chloride hydrochloride
RL: RCT (Reactant)
(reaction of, in prepn. of tocotrienol for teating hypercholesterolemia, hyperlipidiemia and **thromboembolic** disorders)

IT 53254-60-7
RL: RCT (Reactant)
(reaction of, in prepn. of tocotrienol for teating hypercholesterolemia, hypolipidemia and **thromboembolic** disorders)

IT 1983-71-7 1983-71-7D, cyclized 3970-21-6 5717-37-3, Ethyl 2-(triphenylphosphoranylidene)propionate 64218-01-5
RL: RCT (Reactant)
(reaction of, in prepn. of tocotrienol for treating hypercholesterolemia, hyperlipidemia and **thromboembolic** disorders)

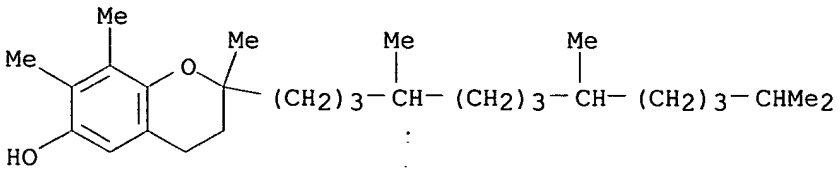
L46 ANSWER 30 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1990:578287 HCAPLUS
DN 113:178287
TI Oral enteric-coated dosage forms of .omega.-3 polyunsaturated fatty acids
IN Pluess, Roger Andre
PA Tillotts Pharma A.-G., Switz.
SO Brit. UK Pat. Appl., 11 pp.
CODEN: BAXXDU
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	GB 2223943	A1	19900425	GB 1988-24709	19881021
	CA 2000881	AA	19900421	CA 1989-2000881	19891017
	WO 9004391	A1	19900503	WO 1989-GB1251	19891020
	W: AU, DK, FI, GB, JP, NO, US				
	RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE				
	AU 8944856	A1	19900514	AU 1989-44856	19891020
PRAI	GB 1988-24709		19881021		
	WO 1989-GB1251		19891020		

AB The title fatty acids, esp. all-cis-5,8,11,14,17-eicosapentaenoic acid (EPA) and/or 22:6 .omega.-3-docosahexaenoic acid (DHA), as free acids or pharmaceutically acceptable salts, are provided in enteric dosage forms; these acids may be optionally used with other active principles, esp. linoleic acid, .gamma.-linolenic acid, and/or dihalo-.gamma.-linolenic acid. Preferably, the enteric dosage form is an enterically coated capsule, e.g. a soft, or esp. hard, gelatin capsule. The dosage form is useful for treatment of a variety of diseases and as a dietetic. Thus, transparent hard gelatin capsules were filled with 500 mg of a fish oil conc. contg. free EPA 32, free DHA 28, and .**gamma**.-**tocopherol** 0.02 wt.%.

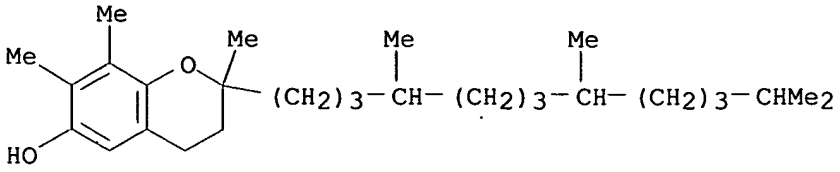
IT **7616-22-0**, .**gamma**.-**Tocopherol**
RL: BIOL (Biological study)
(enteric-coated pharmaceutical contg. .omega.-3 polyunsatd. fatty acid

and)
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



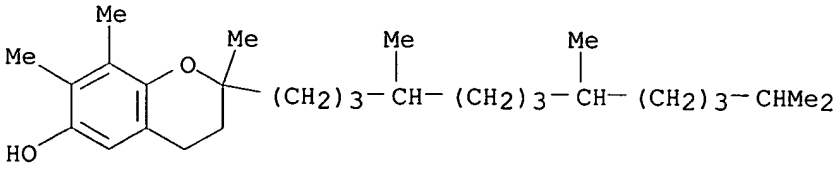
IC ICM A61K031-20
CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1, 18
IT **Embolism**
(**thrombo-**, treatment of, .omega.-3 polyunsatd. fatty acid-contg. enteric-coated pharmaceutical for)
IT 60-33-3, Linoleic acid, biological studies 506-26-3, .gamma.-Linolenic acid 1783-84-2 **7616-22-0, .gamma.-Tocopherol**
RL: BIOL (Biological study)
(enteric-coated pharmaceutical contg. .omega.-3 polyunsatd. fatty acid and)

L46 ANSWER 31 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1988:5036 HCAPLUS
DN 108:5036
TI Vitamin E status in a normal population: the influence of age
AU Vandewoude, Maurits F. J.; Vandewoude, Michel G.
CS Dep. Nutr. Metab., Univ. Antwerp, Wilrijk, B-2610, Belg.
SO J. Am. Coll. Nutr. (1987), 6(4), 307-11
CODEN: JONUDL; ISSN: 0731-5724
DT Journal
LA English
AB Plasma vitamin E and lipids were detd. in 95 healthy volunteers (mean age 55.9 .+-. 24.5 yr). Special attention was focused on vitamin E status in the elderly: 23 individuals were older than 80 yr. A significant age effect was obsd. for both vitamin E and cholesterol, both being increased in the middle-aged group (40-59 yr) and decreased in the elderly (.gtoreq.80 yr). Since a high plasma cholesterol represents a major risk factor for ischemic **heart disease**, decreasing levels of plasma cholesterol with advancing age in a healthy population-sample appears to be the result of neg. selection. Plasma vitamin E concn. was correlated with total cholesterol, triglyceride, and total lipid. Since vitamin E is mainly transported by plasma lipoproteins, these strong correlations suggest that changes in vitamin E should be considered as an epiphenomenon of altered plasma transport capacity. The detn. of plasma vitamin E is therefore a poor indicator of the real tissue vitamin E activity.
IT **7616-22-0, .gamma.-Tocopherol**
RL: BIOL (Biological study)
(of blood plasma, of humans, age effect on)
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 18-2 (Animal Nutrition)
IT 57-88-5, Cholesterol, biological studies 59-02-9, .alpha.-Tocopherol **7616-22-0, .gamma.-Tocopherol**
RL: BIOL (Biological study)
(of blood plasma, of humans, age effect on)

L46 ANSWER 32 OF 32 HCAPLUS COPYRIGHT 2002 ACS
AN 1985:486887 HCAPLUS
DN 103:86887
TI Advances in vitamin E research
AU Machlin, Lawrence J.
CS Hoffmann-La Roche Inc., Nutley, NJ, USA
SO Bitamin (1985), 59(7), 253-61
CODEN: BTMNA7; ISSN: 0006-386X
DT Journal
LA English
AB Men given dl-.alpha.-tocopheryl acetate [52225-20-4] or d-.alpha.-tocopheryl acetate [58-95-7] supplements (800 IU/day) orally showed increased plasma .alpha.-tocopherol [59-02-9] to a plateau at 2,2-2,6 mg/dL by the 3rd-7th day; the dl-form produced slightly greater plasma tocopherol levels. Both of these vitamin supplements produced greatly decreased plasma **.gamma.-tocopherol** [**7616-22-0**] levels. Studies in dogs and rats indicated that although low levels of vitamin E [1406-18-4] (15 mg/kg in rats) are needed to prevent myopathy and testis degeneration, and even lower levels (7.5 mg/kg) to prevent growth retardation, high levels are needed for optimal immune responses. The vitamin E nutrition of premature infants and children and the involvement of vitamin E in platelet function, **heart disease**, and cancer are also discussed.
IT **7616-22-0**
RL: BIOL (Biological study)
(of blood plasma, of humans, dietary tocopherol acetate isomers effect on)
RN 7616-22-0 HCAPLUS
CN 2H-1-Benzopyran-6-ol, 3,4-dihydro-2,7,8-trimethyl-2-(4,8,12-trimethyltridecyl)- (9CI) (CA INDEX NAME)



CC 18-2 (Animal Nutrition)
IT 59-02-9 **7616-22-0**
RL: BIOL (Biological study)
(of blood plasma, of humans, dietary tocopherol acetate isomers effect on)